

The Journal of Thoracic Surgery

Official Organ
The American Association for Thoracic Surgery

Editor
EVARTS A. GRAHAM
St. Louis

Associate Editors
BRIAN BLADES **THOMAS H. BURFORD**
Washington, D. C. St. Louis

ADVISORY EDITORIAL BOARD

JOHN ALEXANDER, Ann Arbor	STUART W. HARRINGTON, Rochester, Minn.
ALFRED BLALOCK, Baltimore	ROBERT M. JAMES, Toronto
EDWARD D. CHURCHILL, Boston	HERBERT C. MAIER, New York
LEO ELOESSER, San Francisco	RUDOLPH MATAS, New Orleans
RICHARD H. MEADE, JR., Grand Rapids	

ADVISORY EDITORIAL BOARD REPRESENTING THE SOCIETY OF THORACIC SURGERY OF
GREAT BRITAIN AND IRELAND

H. MORRISTON DAVIES, RUTHIN, NORTH WALES	R. C. BROCK, LONDON
C. PRICE THOMAS, LONDON	

VOLUME 19
JANUARY, FEBRUARY, MARCH,
APRIL, MAY, JUNE, 1950

ST. LOUIS
THE C. V. MOSBY COMPANY
1950

COPYRIGHT, 1950, BY THE C. V. MOSBY COMPANY

(All rights reserved)

Printed in the
United States of America

*Press of
The C. V. Mosby Company
St. Louis*

The Journal of Thoracic Surgery

Vol. 19

JANUARY, 1950

No. 1

Original Communications

THE SURGICAL TREATMENT OF MITRAL STENOSIS

II. PROGRESS IN DEVELOPING A CONTROLLED VALVULOPLASTIC TECHNIQUE

DWIGHT E. HARKEN, M.D.,* LAURENCE B. ELLIS, M.D.† (BY INVITATION),
AND LEONA R. NORMAN, M.D.‡ (BY INVITATION)
BOSTON, MASS.

THE background of the surgery of mitral stenosis has been reviewed in a previous communication.¹ The earlier thinking and the earlier experiences²⁻¹⁴ reduced the problem to a consideration of the relative significance of mitral stenosis versus regurgitation. There is now evidence that there are various types of regurgitation, some borne much better than others.

The correction of mitral stenosis is no longer directed at the production of insufficiency but at reducing the stenotic block with the greatest possible restoration of valvular function. While much previous work has been based on the concept of exchanging regurgitation for stenosis, it does not follow that this is either desirable or necessary.

At the present time there is no method of determining quantitatively the degree of regurgitation or of stenosis in a given patient. Since stenosis and regurgitation may affect the pulmonary and cardiac dynamics differently, the choice of operation would be greatly influenced by knowledge of these factors. In gaining such specific knowledge and in evaluating the general cardiovascular status it is highly desirable to apply as many objective tests as possible. At present, these objective tests must include cardiac catheterization for the determination of blood flow and intracardiac and pulmonary vascular pressures.

From the Eighth Surgical Service (Thoracic) and the Surgical Research Laboratory, Boston City Hospital; the Surgical Service of the Peter Bent Brigham Hospital; The Heart Institute of the Chelsea Naval Hospital; and the Department of Surgery, Harvard Medical School.

The medical evaluation of patients and cardiac catheterization studies were carried out at the Thorndike Memorial Laboratory, Second and Fourth Medical Services (Harvard), Boston City Hospital; the Cardiovascular Laboratory, Peter Bent Brigham Hospital; and the Department of Medicine, Harvard Medical School.

This study was supported by The Heart Institute of the Sias Laboratories, Brooks Hospital, Brookline, Mass.; and by The Life Insurance Medical Research Fund.

Read at the Twenty-ninth Annual Meeting of The American Association for Thoracic Surgery, New Orleans, La., March 29-31, 1949.

*Assistant Clinical Professor of Surgery, Harvard Medical School; Visiting Surgeon for Thoracic Surgery, Boston City Hospital; Senior Associate in Thoracic Surgery, Peter Bent Brigham Hospital; Thoracic Surgeon, Mount Auburn and Malden Hospitals.

†Assistant Clinical Professor of Medicine, Harvard Medical School; Associate Physician, Thorndike Memorial Laboratory, Boston City Hospital; Assistant Visiting Physician, Boston City Hospital.

‡Research Fellow in Heart Surgery, Heart Institute of Sias Laboratories, Brooks Hospital.

CLASSIFICATION OF PATIENTS WITH RESPECT TO SURGICAL PROCEDURES

In previous communications a preliminary classification of patients with mitral stenosis was offered with respect to their suitability for different operations. This classification has undergone modification, but the original outline follows:

Group A includes patients with a *low resting cardiac output* which increased little or not at all on exercise. Right ventricular failure may or may not have been present. For such patients, the operation of "valvuloplasty" may be helpful since, in these individuals, mitral obstruction is of major importance in their clinical condition.

Group B comprises patients with *normal resting cardiac outputs* that usually increase with exercise. The pulmonary symptoms may be as severe as in the patients in the first group. In these persons mitral regurgitation rather than stenosis may be the predominant factor, or the cardiac output may be maintained only by a high left auricular pressure driving blood through the stenotic mitral valve. For these patients, a beneficial though purely palliative procedure is the surgical production of an interatrial septal defect or the ingenious azygos to pulmonary vein shunt used by Sweet and Bland¹⁰ to decompress the left auricle and the pulmonary venous hypertension, especially at high peaks during periods of strain. Such operations would not be suitable for patients who have had right ventricular failure because the recirculation of blood through this chamber and the pulmonary circulation would place an added burden on the right ventricle. This procedure would also be contraindicated in patients with a low resting cardiac output; in them, too great a proportion of blood might be diverted through the shunt, and the left auricular pressure might be so lowered that the blood flow through the mitral orifice to the periphery would be insufficient to sustain life.

Group C includes patients whose incapacitating symptoms, particularly attacks of pulmonary edema, are associated with *rapid heart action* that cannot be controlled by medical measures. The cardiac output may be normal or low, and the pulmonary artery pressure is always elevated. The patients are not deemed suitable candidates for either of the operations just mentioned because of the severity of their disease. The occasional patient with mitral stenosis who has attacks of severe chest pain, so-called "hypercyanotic angina," may also fall into this group.²⁰ For these patients, a palliative procedure may be the removal of the cardiac sympathetic accelerator and afferent nerves. In two successive operations, the inferior cervical ganglion and the first four or five dorsal sympathetic ganglions are removed from each side through a small cervical incision. The relief afforded is through the production of a slower heart rate or the interruption of pain fibers. It is also possible that some additional direct effect on the pulmonary circulation is produced.

With increase in our knowledge, gained from operative experience and study of a group of twenty-five patients by cardiac catheterization and other

methods, we have modified this classification. It has become apparent by observation of four cases confirmed at operation or autopsy that *high grade mitral stenosis can exist* in patients whose *resting cardiac output* is normal as determined by catheterization. It is quite probable that this resting output is close to the maximum that can be attained. Therefore, definite clinical improvement may take place in patients following the valvuloplastic operation, with significant lowering of the pulmonary arterial pressure, yet with no change in the level of the resting cardiac output. It is evident, therefore, that too much reliance cannot be placed on the cardiac output alone as a guide to operability. There may be functional and structural alterations in the heart and in the pulmonary circulation. Marked diminution of the stroke volume and still further elevation of the pulmonary vascular pressure may be coincident with the tachycardia of exercise or anxiety.

In the present experimental stage of the procedure, the operations under discussion should be considered only in patients with uncomplicated mitral valve disease in whom there is no evidence of a significant degree of involvement of other valves, of the myocardium itself, or of associated disease.

SOME FUNDAMENTAL PRINCIPLES OF MITRAL VALVULOPLASTIC SURGERY

Four surgical principles pertaining to mitral valvuloplasty have been presented¹:

I. The operation should be performed without dislocation of the heart from position of optimum function.

II. The buttonhole opening of the stenotic mitral valve should be approached from the auricular side so that the funnel directs the cutting instrument toward the leaflet margin.

III. Surgical enlargement of the stenotic orifice should be so planned that there is a minimum burden from associated regurgitation ("selective insufficiency") and maximum restoration of valvular function ("valvuloplasty") (Fig. 1).

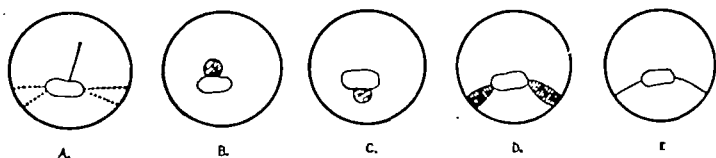


Fig. 1.—Diagrammatic representation of various operations in the stenotic mitral ring as seen from above. A, Anterior valvulotomy (dotted lines indicate zones in the solid stenotic funnel that were commissures in the predisease state); B, anterior leaflet insufficiency; C, posterior leaflet insufficiency—"selective insufficiency"; D, "selective insufficiency" and "valvuloplasty" by excision of the immobilizing commissure bridges; E, "valvuloplasty" by incision in the zones of the commissures.

IV. In the presence of mitral obstruction or regurgitation, undue acceleration of the heart rate must be avoided. This acceleration is avoided by eliminating all possible ordinary sources of tachycardia incident to surgery, by adequate digitalization, and by the use of intravenous procaine at the time of

operation to reduce cardiac irritability. Tachycardia increases pulmonary vascular pressure and is associated with pulmonary edema and other forms of "pulmonary decompensation."

Now we may add two more principles and clarify further some of the previous statements through experience with three new valvuloplasty patients. These case presentations will show that:

V. Blind intracardiac maneuvers must be well controlled and deliberate.

VI. There must be the least possible stimulation of the ventricular endocardium during intracardiac operation.

In this field of surgery, the experimental animal is of only partial help. Each new human problem must be approached with care and the maximum experience gained from each surgical operation. Our intracardiac manipulations have been not only blind, but hurried and ill controlled. It would not be so hazardous to work quickly if we could see well, directly or indirectly. An adequate cardioscope is, unfortunately, not yet available.^{11, 15, 21} Quite naturally, we look forward to the satisfactory extracorporeal pump that will allow deliberate open intracardiac surgery. Björk in Crafoord's laboratory,²² Gibbon,²³ and Jongbloed²⁴ have gone far but they have not yet offered a pump suitable for human use.

It would not be such a handicap to work blindly if one could be deliberate and carefully palpate for the stenotic opening with suitable instruments, could map the pattern of the rolled "fishmouth," and could then deliberately incise or resect the fused commissures, and excise suitable portions of the posterior leaflet. This should be controlled by frequent auricular and ventricular pressure recordings.

Four new operative experiences with valvuloplasty are presented here because they illustrate (a) the need for more deliberate cardiac manipulation, (b) the need for better control of the open auricle and consequent loss of blood, (c) the need for frequent pressure soundings as a guide to the pattern and extent of excision, and (d) the steps taken to meet these needs.

CASE REPORTS

Mitral Stenosis Case 6, Valvuloplasty No. 3.—F. M., a 32-year-old housewife, was referred to the U. S. Naval Hospital, Chelsea, Mass. on Dec. 12, 1948, with the chief complaint of increasing dyspnea for the past seven years.

Seven years previously, during her only pregnancy, a heart murmur was discovered and digitalis was begun. Four years prior to admission, there was an aggravation of symptoms of dyspnea, cough, and frothy sputum. Digitalis was discontinued because of nausea. One year prior to this admission, the patient had an episode of severe cough accompanied by a large quantity of blood-tinged, frothy sputum which was diagnosed as "inter-lobar pleurisy" and subsided with conservative therapy. During the past year, dyspnea, weakness, and marked palpitation of the heart became more distressing. The patient had to restrict her activities greatly. She walked little and had to be carried upstairs. Digitalis was begun again. There has never been any peripheral edema.

The past history revealed no definite history of rheumatic fever. The patient had two attacks of diphtheria about eleven years prior to admission.

Physical examination revealed a pale, thin, rather underdeveloped woman. General physical examination was not remarkable except for the cardiovascular system. The maximal apical impulse was 9 cm. to the left of the midsternal line in the fifth interspace and the

left border of dullness was percussed at 10 cm., 3 cm. beyond the midclavicular line. There was an apical diastolic thrill. The first mitral sound was snapping in quality. There was a Grade II apical systolic and a low-pitched Grade III diastolic rumble with presystolic accentuation as well as a Grade I high-pitched early diastolic murmur in the pulmonic area. The rhythm was regular with a resting rate of 88 that rose to 140 on mild exercise. The lungs were clear. There was no venous congestion, hepatomegaly, ascites, or peripheral congestion. Blood pressure was 120/80.

Teleroentgenography showed a 25 per cent enlargement in the transverse diameter of the heart, and marked prominence of the pulmonary conus with evidence of left auricular enlargement in the oblique view. The electrocardiogram was consistent with right ventricular and auricular enlargement. The venous pressure was 95 to 110 mm. of water, and rose to 140 with hepatic pressure. Blood examination on admission revealed the hemoglobin to be 11 Gm., the red count to be 4,540,000 and hematocrit 41.

Preoperative catheterization studies are shown in Table I.

Mitral valvuloplasty was done Jan. 6, 1949. Premedication was Seconal 100 mg., scopolamine 0.05 mg. Anesthesia was brief pentothal induction, then endotracheal ether.

Electrocardiographic tracings were followed throughout the procedure by medical consultants.

Incision was made from the second rib on the left immediately lateral to the sternum in the inframammary line to the midaxilla. During the opening of the chest wall, electrocardiograms revealed idioventricular rhythm and irregular runs of auricular fibrillation. Extrasystoles and ventricular tachycardia were noted, and on three occasions over a period of thirty minutes, 20 c.c. of 1 per cent procaine were administered intravenously. After reflecting the left breast upward, the incision was carried through the left third interspace from the sternum to the midaxillary line. The mammary vessels were ligated proximally and distally, then divided. As the ribs were exposed, the third and fourth costal cartilages were divided 1.5 cm. to the left of the sternum. Irregularity of beat was accentuated with the spreading of the ribs. The pericardium was opened over the left auricular appendage in a linear fashion 1.5 cm. anterior to the left phrenic nerve, and No. 2 braided silk stay sutures were placed for exposure. As one stay suture was inserted, the auricular appendage was inadvertently nicked, but bleeding was controlled with a small snap.

A 000 black Deknatel suture was inserted in purse-string fashion about the tip of the left auricular appendage and a No. 2 black silk braided suture was set, but not tied at the base of the appendage. At this point in the operation the patient's blood pressure became imperceptible and there was dilatation of the ventricles with runs of ventricular tachycardia, followed by slow idioventricular rhythm and then fibrillation. The heart appeared to be motionless. Manual massage of the heart was instituted. This cardiac massage was carried out by the surgeon (D. E. H.) who placed his left hand palm upward on the phrenic surface of the heart, meanwhile supporting the dilated exposed anterior surface of the heart with the cupped palm of the right hand. After approximately eight minutes, the heart appeared to fibrillate. Throughout this period of apparent arrest slow fibrillation was recorded by the electrocardiography (Fig. 2). Inasmuch as 20 mg. of procaine had been given twelve and three minutes before the episode of cardiac arrest, it was thought possible that the arrest was due to an abnormal response to a small dose of the drug, therefore it was not administered again but rather Neo-synphrine approximately 1 c.c. within the chamber of the right ventricle, and approximately 0.5 c.c. into the wall of each ventricle, followed within fifteen seconds by ventricular dissociation, then by the resumption of relatively normal beats at 88 a minute, and blood pressure 50/40, after 4 gr. of intravenous aminophylline. The patient seemed to stabilize with a blood pressure of 80/60 with a ventricular rate of 125-200. It was decided to carry on with the planned procedure, since it seemed unlikely that the patient would survive unless mechanical improvement was afforded the heart. The punch valvulotome (Fig. 3) was inserted without recovery of tissue; in doing this the auricular appendage was torn and there was considerable blood loss. The reverse scissors type of valvulotome (Fig. 4) was next inserted and incision with the cutting blade carried through the medial commissure. Again there was marked blood loss through the open appendage. The total blood loss was approximately 1,500 c.c. Hemostasis was extremely difficult by digital compression but control was

obtained and a No. 3 silk tie was secured about the base of the appendage. The pericardium was loosely closed. The chest wall closure was completed in layers with interrupted 000 black silk sutures. At the time the skin was closed, the patient had a blood pressure of 80/60 and a pulse rate of 140. Three and one-half hours later, the pulse was 100 and the blood pressure 110/60. At this point, the electrocardiogram resembled those taken before the operation.

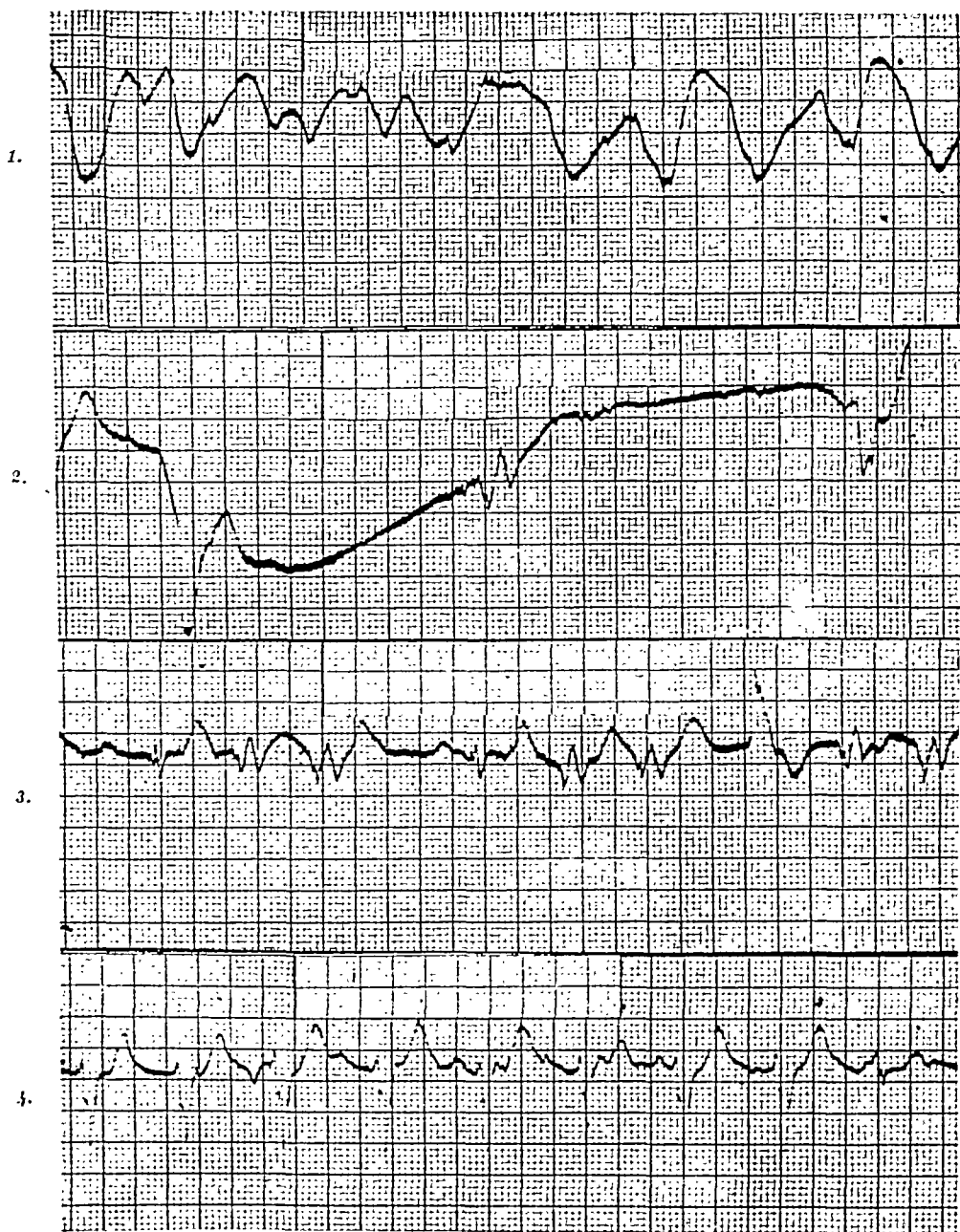


Fig. 2.—Electrocardiographic tracings during valvuloplasty No. 2. From above downward they show (1) bizarre ventricular activity due to slow ventricular fibrillation and the mechanical effect of cardiac massage; (2) very slow bizarre ventricular complexes; (3) idioventricular rhythm with several foci of impulse formation and possible auricular activity; (4) idioventricular rhythm or auriculoventricular nodal rhythm and intraventricular block with apparent auriculoventricular dissociation.

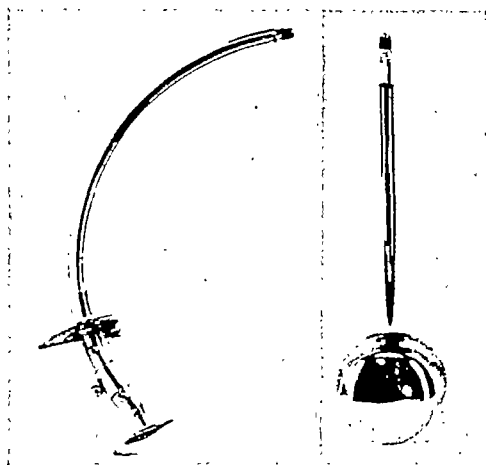


Fig. 3.—The cardiovalvulotome of the punch type.

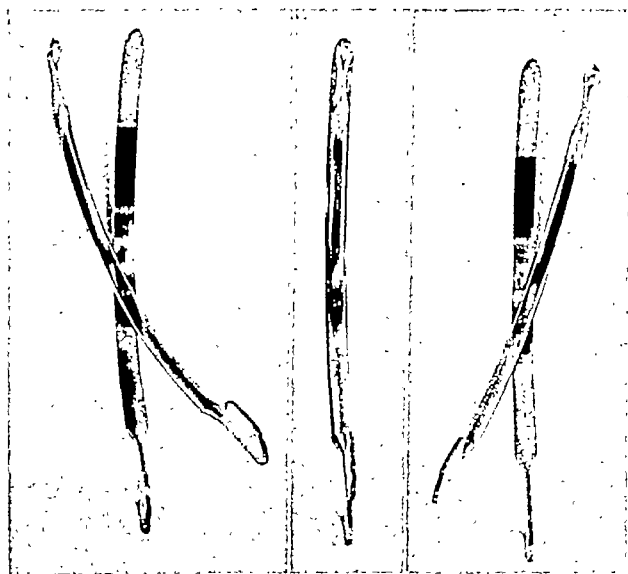


Fig. 4.—The reverse scissors type of valvulotome. The upper blade is the cutting blade. The lower guard blade affords opposing counterpressure for cutting in either direction.

The postoperative course was surprisingly uneventful. There was a marked change in the character of the apical cardiac murmur to a harsh systolic and a faint, short diastolic without presystolic accentuation.

The patient was able to be up and about the ward on the eighth day after operation and even then felt able to perform greater physical effort than before the operation. She was discharged from the hospital on February 12. Two months after operation, she was able to climb stairs for the first time in two years and to perform graded exercises with much less dyspnea.

Postoperative catheterization studies are shown in Table I.

TABLE I. F. M. (MITRAL STENOSIS CASE 6, VALVULOPLASTY NO. 3)

	CARDIAC INDEX	STROKE VOLUME (C.C. PER BEAT)	PULMONARY ARTERY PRESSURE (MM. HG)		
			SYSTOLIC	DIASTOLIC	MEAN
Preoperative (12/28/48)					
Resting	2.43	50.0	77.5	30.5	49
After exercise	2.92	36.5	122.5	64.5	74
Postoperative (3/16/49)					
Resting	2.84	46.5	50.0	21.5	32
After exercise	3.18	49.0	73.8	31.5	46

Comment.—This record is significant for several reasons. Not only can an extremely ill patient survive mitral valvuloplasty, but there is unmistakable objective evidence of improvement following this operation. Survival alone is not a sufficient assay of surgical success.

There are perhaps only two previous examples of ventricular fibrillation on the operating table, proved by electrocardiography, that have reverted to effective beat coincident with maneuvers on the part of the surgeon directed at defibrillation of the heart (Beek²⁵ and Lampson²⁶).

Finally, the lack of control of blood loss from the auricle after it was torn emphasized the grave need for more dependable means of grasping and fixing this structure as well as more deliberate hemostatic means of introducing instruments into the heart.

Mitral Stenosis Case 7, Valvuloplasty No. 4.—E. C., a 36-year-old white married woman, entered the Boston City Hospital Feb. 11, 1949. This was her ninth hospital admission with chief complaint of hemoptysis.

Thirteen years prior to this admission, she first noted bloodstreaked sputum, with increasing fatigability. Several months after the onset of symptoms, she had a massive hemoptysis, at which time she was told she had serious heart disease. There was no history of acute rheumatic fever. Digitalis was begun at that time, and has been continued since. Since the initial episode, she has had recurrent bloody sputum, difficulty in breathing, and has led the life of an invalid at home and in hospitals. Her hospital admissions were necessitated by episodes of pulmonary edema.

Since the last hospitalization one year before admission, she has suffered dyspnea on walking a few feet, marked orthopnea, frequent attacks of paroxysmal nocturnal dyspnea, and mild intermittent ankle edema.

On the night before this admission, the patient vomited several times, noted tachycardia with palpitation, and gradual increase in dyspnea accompanied by frothy pink sputum. She was admitted in acute pulmonary edema with evidence of a pulmonary infection. She responded to therapy, including oxygen, sedatives, antibiotics, and diuretics.

Physical examination when she was convalescent two weeks later was as follows: Her appearance was that of an emaciated 30-year-old woman. The general physical examination was not remarkable except for the cardiovascular examination. The lungs were clear. The heart was enlarged to the left, and there was no evidence of peripheral congestion. There was a diffuse precordial thrust with an apical presystolic thrill. Over the mitral area was heard a diastolic rumble with crescendo presystolic murmur and a loud mitral first sound. The second pulmonic sound was loud and split. The rhythm was regular, heart rate 98. Blood pressure was 100/54.

Teleroentgenograms and fluoroscopy revealed marked enlargement of the pulmonary artery at the conus, increased arterial markings in both lung fields, and enlargement in the transverse diameter. Electrocardiograms gave evidence of right ventricular enlargement. The hematocrit was 40, the hemoglobin 14 Gm. The white blood cell count was 7,350.

Cardiac catheterization showed the cardiac output to be 4.57 liters per minute (cardiac index, 3.06) with no appreciable change in output or stroke volume on exercise. Pulmonary artery pressures were not obtained.

Mitral valvuloplasty was done March 14, 1949. Premedication was Seconal 0.40 Gm., demerol 100 mg., scopolamine 0.65 mg. Anesthesia was cyclopropane induction, then endotracheal ether.

Electrocardiographic tracings were followed throughout.

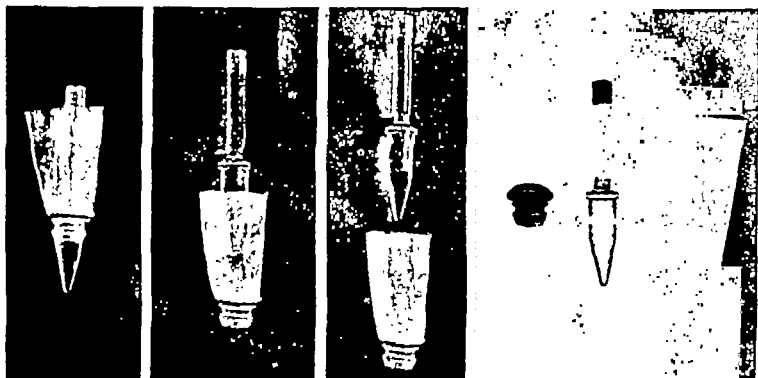


Fig. 5.—The operating tunnel with introducing obturator and plastic control sleeve.

With the patient in the dorsal decubitus position, a curved 34 cm. incision was carried from the left sternal border below the left breast to the anterior axillary line. The breast was reflected upward and the chest opened in the third intercostal space. The costal cartilages of the third and fourth ribs were divided 1.5 cm. to the left of the sternum and the ribs spread. The pericardium was opened anterior to the phrenic nerve and silk marsupializing traction sutures were placed. A greatly enlarged pulmonary artery measuring perhaps 6 cm. in diameter presented and caused cardiac rotation that gave the left auricular appendage a posterior position. This position of the appendage necessitated a second incision posterior to and parallel to the phrenic nerve, and thus afforded adequate appendage exposure. The margins of the auricular appendage were grasped by two Kelly clamps, the auricle between the clamps was incised, and the operating tunnel inserted (Fig. 5). A silk suture of No. 2 silk previously laid at the base of the appendage was then tied, but was not properly placed and the polythene cuff of the tunnel occluded. Efforts at using the well were abandoned. There had been little blood loss and the patient's condition remained good.

The curved valvulotome punch was then directly introduced with considerable blood loss (perhaps 1000 c.c.). Two bites of the mitral valve were taken. The blood pressure fell rapidly, but the heartbeat remained regular. Pressure transfusion brought the blood pressure to normal levels within ten minutes. It was felt that the valvular excision had not been adequate so the valvulotome was reintroduced and another bite taken. This was again accompanied by marked blood loss, at least 1,500 c.c., and the patient went into shock.

Although there was marked bleeding during the intracardiac manipulation, it was twice demonstrated that the auricle clamp (Fig. 6) worked nicely in controlling blood loss between manipulations. During and following these manipulations 3000 c.c. of blood were delivered under pressure, but we did not feel that the rapid replacement technique was as satisfactory as it might have been.

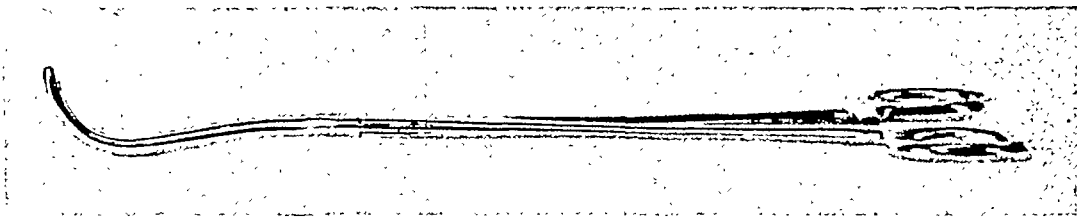


Fig. 6.—The auricle clamp—a noncrushing clamp with teeth to prevent slipping.

With the onset of shock, the heartbeat became irregular, there was auriculoventricular block recorded by continuous electrocardiogram, then idioventricular rhythm, and finally ventricular arrest and fibrillation. Cardiac massage was instituted, positive pressure respiration conducted, Neo-synephrine injected intracardially, and finally, electric shock applied. All measures were futile. Massage and artificial respiration were continued for thirty minutes without resumption of autogenous activity. Permission for post-mortem examination was not obtained.

Comment.—This case illustrates the devastating effects of hurried, blind operating with inadequate control of blood loss. The auricular incision and therefore subsequent blood loss was much larger than in any other operation because the well had been inserted and removed. Doubtless the operating well should not have been abandoned after one simple initial complication in insertion. Certainly in retrospect we can say that the second intracardiac maneuver should not have been attempted.

Mitral Stenosis Case 8, Valvuloplasty No. 5.—M. H., a 29-year-old male veteran, was admitted to the Peter Bent Brigham Hospital on Feb. 21, 1949, for evaluation of his cardiac status.

Seven years previously, in November, 1942, he was refused admission to Officer-Candidate School because of a "presystolic murmur." Army discharge followed shortly. The patient had few complaints until years later. In 1945, he began to suffer from pains over the precordium associated with dyspnea that was frequently asthmatic in character. These increased in severity over the next two years. Late in 1946, hemoptysis occurred and varied from streaking to frank hemorrhage of 750 c.c. At this time the patient was admitted to a Veterans Administration Hospital, where he was observed for nine months. During the course of this hospitalization there were occasional episodes of bleeding. At times he had a low-grade fever and lesions suspected of being petechial in character were noted. The patient was digitalized at this time and has continued on 0.2 mg. of digitoxin daily since. He was given ammonium chloride, Mercuhydrin, and salicylates. Following his discharge from that hospital in September, 1947, he was fairly comfortable for a few weeks until pain, dyspnea,

The curved valvulotome punch was then directly introduced with considerable blood loss (perhaps 1000 c.c.). Two bites of the mitral valve were taken. The blood pressure fell rapidly, but the heartbeat remained regular. Pressure transfusion brought the blood pressure to normal levels within ten minutes. It was felt that the valvular excision had not been adequate so the valvulotome was reintroduced and another bite taken. This was again accompanied by marked blood loss, at least 1,500 c.c., and the patient went into shock.

Although there was marked bleeding during the intracardiac manipulation, it was twice demonstrated that the auricle clamp (Fig. 6) worked nicely in controlling blood loss between manipulations. During and following these manipulations 3000 c.c. of blood were delivered under pressure, but we did not feel that the rapid replacement technique was as satisfactory as it might have been.

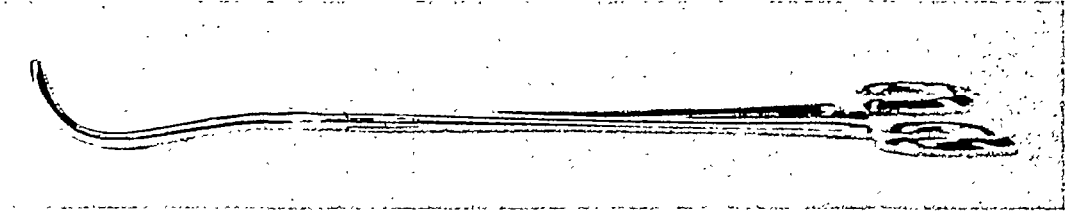


Fig. 6.—The auricle clamp—a noncrushing clamp with teeth to prevent slipping.

With the onset of shock, the heartbeat became irregular, there was auriculoventricular block recorded by continuous electrocardiogram, then idioventricular rhythm, and finally ventricular arrest and fibrillation. Cardiac massage was instituted, positive pressure respiration conducted, Neo-syneprine injected intracardially, and finally, electric shock applied. All measures were futile. Massage and artificial respiration were continued for thirty minutes without resumption of autogenous activity. Permission for post-mortem examination was not obtained.

Comment.—This case illustrates the devastating effects of hurried, blind operating with inadequate control of blood loss. The auricular incision and therefore subsequent blood loss was much larger than in any other operation because the well had been inserted and removed. Doubtless the operating well should not have been abandoned after one simple initial complication in insertion. Certainly in retrospect we can say that the second intracardiac maneuver should not have been attempted.

Mitral Stenosis Case 8, Valvuloplasty No. 5.—M. H., a 29-year-old male veteran, was admitted to the Peter Bent Brigham Hospital on Feb. 21, 1949, for evaluation of his cardiac status.

Seven years previously, in November, 1942, he was refused admission to Officer-Candidate School because of a "presystolic murmur." Army discharge followed shortly. The patient had few complaints until years later. In 1945, he began to suffer from pains over the precordium associated with dyspnea that was frequently asthmatic in character. These increased in severity over the next two years. Late in 1946, hemoptysis occurred and varied from streaking to frank hemorrhage of 750 c.c. At this time the patient was admitted to a Veterans Administration Hospital, where he was observed for nine months. During the course of this hospitalization there were occasional episodes of bleeding. At times he had a low-grade fever and lesions suspected of being petechial in character were noted. The patient was digitalized at this time and has continued on 0.2 mg. of digitoxin daily since. He was given ammonium chloride, Mercuhydrin, and salicylates. Following his discharge from that hospital in September, 1947, he was fairly comfortable for a few weeks until pain, dyspnea,

this incision the operating tunnel was inserted. The auricular wall was tied in place with a No. 5 black silk suture. Little blood was lost during this maneuver.

The curved punch valvulotome was introduced through the operating tunnel, the mitral fishmouth located, and two substantial cuts were made in a region taken to be that of the posterior leaflet. Segments of valve leaflet on the zones of fused commissures were not removed in the valvulotome. The valvulotome was introduced twice. The second time the punch was inserted tiny fragments of tissue resembling bits of chordae tendineae were found in the instrument. A No. 5 black silk tie was then placed about the base of the appendage and the suprajacent portion was cut off to recover the tunnel. At this point, the blood pressure had fallen beyond detection and the heart action became so feeble as to suggest impending arrest. However, after a rest period of five minutes the action had recovered sufficiently that a blood pressure of 80/40 was obtained and the continuous electrocardiogram showed a regular rate of 104 beats a minute.

The pressure in the auricle was not taken because the trocar used for such soundings had perforated the plastic sleeve of the tunnel and also had damaged the wall of the left auricle. Both of these perforations resulted in blood loss that made the intracardiac maneuvers less than completely deliberate. Blood loss through the laceration of the auricle was controlled by pressure with an Oxycel sponge and the sleeve perforation was occluded by digital compression until the auricular appendage had been tied and amputated.

The pericardium was then loosely closed, leaving the Oxycel sponge on the auricle at the point of perforation.

Approximately 1,200 c.c. of blood were aspirated from the left pleural space, the lung was expanded, and 400,000 units of penicillin were instilled. The chest wall was closed with interrupted silk sutures, without drainage.

The left auricular appendage removed at operation showed histologic changes consistent with an active rheumatic carditis. These included perivascular foci of oval vesicular cells believed to represent Aschoff bodies.

The postoperative course was moderately stormy, due primarily to the patient's limited respiratory reserve, to great apprehension attending discomfort, and to considerable bloody effusion in the left pleural space.

After four days of apparent improvement, he suddenly went into pulmonary edema and died within a few hours in spite of standard therapy.

Gross autopsy findings were essentially those of far-advanced stenosis with the pulmonary vascular changes that may be associated with this condition. The valvulotome had produced a defect 3 by 1 mm. in the medial commissure and a much smaller defect, virtually a scratch of the lateral commissure. The classical marginal ring at the vortex of the funnel that prevents mitral valve opening or closure had not been broken. The stenosis had not been corrected by valvuloplasty, nor had it been significantly altered by "selective insufficiency."

Comment.—The operative and autopsy findings in this patient re-emphasize some cardinal features of the pathology and surgical technique in the correction of mitral stenosis.

The operating well was used successfully here; it may make intracardiac maneuvers more deliberate, with better control of blood loss. We look forward to using the operating tunnel as the portal through which damaged valves may be excised and prosthetic valves be inserted. Hufnagel is now making significant progress in the use of plastic valves.²⁷

In this operation, alarming evidence of ventricular irritability and disturbances of electrical conduction developed when the ventricular endocardium was stimulated. This has become apparent by correlating our direct observations of the heart at operation with the continuous electrocardiogram, and by repeated observations during cardiac catheterization.

this incision the operating tunnel was inserted. The auricular wall was tied in place with a No. 5 black silk suture. Little blood was lost during this maneuver.

The curved punch valvulotome was introduced through the operating tunnel, the mitral fishmouth located, and two substantial cuts were made in a region taken to be that of the posterior leaflet. Segments of valve leaflet on the zones of fused commissures were not removed in the valvulotome. The valvulotome was introduced twice. The second time the punch was inserted tiny fragments of tissue resembling bits of chordae tendineae were found in the instrument. A No. 5 black silk tie was then placed about the base of the appendage and the suprajacent portion was cut off to recover the tunnel. At this point, the blood pressure had fallen beyond detection and the heart action became so feeble as to suggest impending arrest. However, after a rest period of five minutes the action had recovered sufficiently that a blood pressure of 80/40 was obtained and the continuous electrocardiogram showed a regular rate of 104 beats a minute.

The pressure in the auricle was not taken because the trocar used for such soundings had perforated the plastic sleeve of the tunnel and also had damaged the wall of the left auricle. Both of these perforations resulted in blood loss that made the intracardiac maneuvers less than completely deliberate. Blood loss through the laceration of the auricle was controlled by pressure with an Oxyel sponge and the sleeve perforation was occluded by digital compression until the auricular appendage had been tied and amputated.

The pericardium was then loosely closed, leaving the Oxyel sponge on the auricle at the point of perforation.

Approximately 1,200 c.c. of blood were aspirated from the left pleural space, the lung was expanded, and 400,000 units of penicillin were instilled. The chest wall was closed with interrupted silk sutures, without drainage.

The left auricular appendage removed at operation showed histologic changes consistent with an active rheumatic carditis. These included perivascular foci of oval vesicular cells believed to represent Aschoff bodies.

The postoperative course was moderately stormy, due primarily to the patient's limited respiratory reserve, to great apprehension attending discomfort, and to considerable bloody effusion in the left pleural space.

After four days of apparent improvement, he suddenly went into pulmonary edema and died within a few hours in spite of standard therapy.

Gross autopsy findings were essentially those of far-advanced stenosis with the pulmonary vascular changes that may be associated with this condition. The valvulotome had produced a defect 3 by 1 mm. in the medial commissure and a much smaller defect, virtually a scratch of the lateral commissure. The classical marginal ring at the vortex of the funnel that prevents mitral valve opening or closure had not been broken. The stenosis had not been corrected by valvuloplasty, nor had it been significantly altered by "selective insufficiency."

Comment.—The operative and autopsy findings in this patient re-emphasize some cardinal features of the pathology and surgical technique in the correction of mitral stenosis.

The operating well was used successfully here; it may make intracardiac maneuvers more deliberate, with better control of blood loss. We look forward to using the operating tunnel as the portal through which damaged valves may be excised and prosthetic valves be inserted. Hufnagel is now making significant progress in the use of plastic valves.²⁷

In this operation, alarming evidence of ventricular irritability and disturbances of electrical conduction developed when the ventricular endocardium was stimulated. This has become apparent by correlating our direct observations of the heart at operation with the continuous electrocardiogram, and by repeated observations during cardiac catheterization.

The microscopic finding of active rheumatic carditis points out the need for clinical tests for assessing disease activity. By our own criteria, we should not have selected this patient for surgery had we been able to predetermine the presence of such activity.

At autopsy, it was revealed that the mitral valve did not have a diffuse calcific thickening of the fishmouth funnel. Most of the patients with mitral stenosis that we have seen at autopsy have not had such calcification. They have had thickened leaflets, to be sure, but large areas of the leaflets have been thin to the point of translucency and quite flexible. This cannot be readily appreciated in specimens after fixation in preservative. The significant point in stenosis is rather the marked thickening in a *ring* of dense fibrous tissue at and just back of the free margin. This fibrous ring is continuous across the fused commissural zones. At the point of the fused commissures *this ring prevents the closure of the valve in systole*, just as it prevents opening of the valve in diastole.

It appears to us that we can expect considerable improvement in the valvular action of the thickened mitral funnel if this ring is broken, preferably by incision or wedge resection in both commissural zones (Fig. 1). This allows some restoration of function to the thickened leaflets. Obviously, the thickening and shortening of the chordae tendineae, the papillary muscles, and the leaflets themselves may preclude the restoration of perfect valvular function.

This concept of the nature of the mechanical difficulty in mitral stenosis throws considerable light on the reason for the high mortality in the surgery of the disease. If this sort of major surgery is performed without correction of the mechanical handicap, a *properly selected* patient is in no condition to withstand the operation. Conversely, it would appear that patients can withstand valvuloplasty if it does properly improve the underlying valvular disease. The second and third patients in the valvuloplasty series, who were the gravest of risks and operated upon during a period of pulmonary edema, demonstrated this fact.

DISCUSSION

Our experience with mitral stenosis now involves a study group of twenty-five patients, eight of whom have been treated surgically. There have been three surgical deaths. The operations have been as follows: Five valvuloplasties, two interatrial septal defects, and one denervation. The deaths were all in the valvuloplasty group. The first patient of the series was lost because we did not at that time appreciate the devastating effect of tachycardia. The second death was the fourth valvuloplasty. This death was on the operating table from uncontrolled blood loss. The third death was from pulmonary edema on the fifth postoperative day, and autopsy revealed that too little valvuloplastic incision had been made. This debilitated patient was therefore subjected to a very strenuous operation without the relief of adequate correction of the fundamental mechanical handicap.

The experience to date seems to indicate that essential knowledge of the technical difficulties and the indications for various surgical procedures is being gathered.

The possibility of practical surgical relief of mitral stenosis calls for more complete understanding of the disease. Means must be sought to predict the probable clinical course of a given patient. Those who have a truly malignant form of valvular disease should be selected, if possible, before the pulmonary changes render the patient an inordinately serious risk for surgical intervention.

Objective evaluation before, during, and after operation must be conducted in order to determine which procedures are best tolerated and most beneficial. These appraisals should include detailed observations of the cardiac and pulmonary dynamics, both at rest and during exercise.

SUMMARY

1. A tentative classification of patients suffering from mitral stenosis and the operations considered appropriate in those conditions is discussed.

2. Three additional patients who have had valvuloplastic operations have been presented.

3. One of the new patients has completed postoperative evaluation and shows conclusive improvement associated with the valvuloplastic operation.

4. The total experience with 25 patients in the study group has been mentioned in relation to 8 patients who have had surgical treatment. The 8 patients have had the following operations: 5 valvuloplastic procedures, 2 shunts through the provision of septal defects, and 1 cardiac denervation. There were 3 surgical deaths, all following valvuloplasty.

5. An operating tunnel is presented. This tunnel provided an opportunity for more deliberate, better-controlled intracardiac operation in a human valvuloplasty.

6. The mechanical problem posed by the thick marginal ring of fibrous tissue seen in mitral stenosis is reviewed.

7. Two additional principles of mitral valvuloplastic surgery have been presented:

V. Blind intracardiac maneuvers must be well controlled and deliberate.

VI. There must be least possible stimulation of the ventricular endocardium during intracardiac operation.

We wish to express our appreciation for criticisms and suggestions to the following members of the Board of Consultants of The Heart Institute of the Sias Laboratories: Drs. William S. Derrick, Lewis Dexter, Burton E. Hamilton, Francis D. Moore, Frederic Parker, Jr., Richard Schatzki, and Paul M. Zoll.

Appreciation is also expressed to Codman & Shurtleff, Inc., 104 Brookline Avenue, Boston, Mass., for their cooperation in designing and producing these instruments.

REFERENCES

1. Harken, D. E., Ellis, L. B., Ware, P. F., and Norman, L. R.: The Surgical Treatment of Mitral Stenosis. I. Valvuloplasty, *New England J. Med.* 239: 801-809, 1948.
2. Ellis, L. B., and Harken, D. E.: Mitral Stenosis; Clinical Physiology Correlated With Special Reference to Surgical Intervention, *Proc. Am. Clin. and Climat. Assoc.* (In press.)
3. Brunton, L.: Preliminary Note on Possibility of Treating Mitral Stenosis by Surgical Methods, *Lancet* 1: 352, 1902.
Idem: Surgical Operation for Mitral Stenosis, *Ibid.* 1: 547, 1902.

4. Haecker, R.: Experimentelle Studien zur Pathologie und Chirurgie der Herzens, Arch. f. klin. Chir. 84: 1035-1098, 1907.
5. Bornheim, B. M.: Experimental Surgery of Mitral Valve, Bull. Johns Hopkins Hosp. 20: 107-110, 1909.
6. Cushing, H., and Branch, J. R. B.: Experimental and Clinical Notes on Chronic Valvular Disease in Dog and Possible Relations to Future Surgery of Cardiac Valves, J. Med. Research 12: 471-486, 1907-1908.
7. Schepelmann, E.: Versuche zur Herzchirurgie, Arch. f. klin. Chir. 97: 739-751, 1912.
Idem: Herzklappen Chirurgie, Experimentelle Untersuchungen, Deutsche Ztschr. f. Chir. 120: 562-579, 1913.
8. Carrel, A., and Tuohler, T.: Etude anatomopathologique et experimentale sur la chirurgie des orifices du coeur, Presse med. 22: 173-177, 1914.
9. Cutler, E. C., and Levine, S. A.: Cardiomy and Valvulotomy for Mitral Stenosis. Experimental Observations and Clinical Notes Concerning Operated Case With Recovery, Boston M. & S. J. 188: 1023-1027, 1923.
10. Cutler, E. C., Levine, S. A., and Beck, C. S.: Surgical Treatment of Mitral Stenosis: Experimental and Clinical Studies, Arch. Surg. 9: 689-821, 1924.
11. Beck, C. S., and Cutler, E. C.: Cardiovalvulotomy, J. Exper. Med. 40: 375-379, 1924.
12. Cutler, E. C.: Surgical Aspect of Mitral Stenosis, Arch. Surg. 12: 212-220, 1926.
13. Cutler, E. C., and Beck, C. S.: Surgery of Heart and Pericardium, Chap. IV, In Nelson's Loose-Leaf Living Surgery, Vol. 4, edited by A. O. Whipple and F. B. St. John, New York, 1927, Thor. Nelson & Sons, pp. 286-320.
14. Cutler, E. C., and Beck, C. S.: Present Status of Surgical Procedures in Chronic Valvular Disease of Heart: Final Report of All Surgical Cases, Arch. Surg. 18: 403-416, 1929.
15. Allen, D. S., and Graham, E. A.: Intracardiac Surgery—New Method: Preliminary Report, J. A. M. A. 79: 1028-1030, 1922.
16. Allen, D. S.: Intracardiac Surgery, Arch. Surg. 8: 317-320, 1924.
17. Souttar, H. S.: Surgical Treatment of Mitral Stenosis, Brit. M. J. 2: 603-606, 1925.
18. Pribram, B. O.: Die operative Behandlung der Mitralstenose, Arch. f. klin. Chir. 142: 458-465, 1926.
19. Bland, E., and Sweet, R. H.: Personal communication.
Idem: (Cases From the Medical Grand Rounds, Massachusetts General Hospital, Case 53). A Venous Shunt for Marked Mitral Stenosis, Am. Pract. 2: 756-761, 1948.
20. Burgess, A. M., Jr., and Ellis, L. B.: Chest Pain in Patients With Mitral Stenosis, With Particular Reference to So-Called "Hypercyanotic Angina," New England J. Med. 226: 937-941, 1942.
21. Harken, D. E., and Glidden, E. M.: Experiments in Intracardiac Surgery. II. Intracardiac Visualization, J. THORACIC SCRO. 12: 566-572, 1943.
22. Björk, V. O.: Brain Perfusion in Dogs With Artificially Oxygenated Blood, Acta chir. Scandinav. 96 (Suppl. 137), 1948.
23. Gibbon, J. H., Jr.: Maintenance of Life During Experimental Occlusion of the Pulmonary Artery Followed by Survival, Surg., Gynec. & Obst. 69: 602-614, 1939.
24. Jongbloed, J.: A Mechanical Heart and Lung, Nederl. tijdschr. v. geneesk. 92: 1005, 1949; Abstracted in J. A. M. A. 138: 48-49, 1949.
25. Beck, C. S., Pritchard, W. H., and Feil, H. S.: Ventricular Fibrillation of Long Duration Abolished by Electric Shock, J. A. M. A. 135: 985-986, 1947.
26. Lampson, R. S., Schaeffer, W. C., and Lincoln, J. R.: Acute Circulatory Arrest From Ventricular Fibrillation for Twenty-Seven Minutes With Complete Recovery, J. A. M. A. 137: 1575-1578, 1948.
27. Hufnagel, C. A.: Personal communication.

(See page 45 for discussion)

THE SURGERY OF MITRAL STENOSIS

CHARLES P. BAILEY, M.D., ROBERT P. GLOVER, M.D. (BY INVITATION), AND
THOMAS J. E. O'NEILL, M.D. (BY INVITATION)
PHILADELPHIA, PA.

IT WAS inevitable that the many recent advances in cardiac diagnosis and surgery would shortly foster invasion of the heart chambers. Whereas cardiac surgery for the most part has been the surgery of the great vessels encompassing the heart and as such might better have been termed cardiovascular surgery, recent intracardiac invasion has re-affirmed its right as a definitive entity. The eventual scope of such endeavor is limited only by the anatomical boundaries of the organ itself, and by the enlightened intrepitude of its invaders.

It is logical that chronic valvular disease of the heart should be among the first intracardiac disorders subjected to surgical attack. It is common and found universally. White¹ has estimated that 0.5 to 1 per cent of the community is affected, particularly in those areas where rheumatic fever is endemic such as in the northeastern part of the United States and Northern Europe. The great majority of persons who have had rheumatic fever (about 85 per cent) develop deforming valvular disease of the heart, the mitral valve being most frequently affected. Organic stenosis of the mitral valve is probably the commonest single end result of rheumatic infection, although organic aortic valve disease in the form of a regurgitant lesion is rated an equal by some.

The prognosis of an individual case of mitral stenosis depends upon many factors: the age of the patient, the severity of the lesion, the presence or absence of other valve defects, the presence or absence of additional rheumatic activity, and the condition of the myocardium. The ultimate outcome, however, is almost invariably unfavorable. Not only is the life span shortened, but handicapped activity, invalidism, and occasionally vegetation are present among its victims. Although a few may escape until later years, it is more often fatal before or soon after the patient reaches adult life. Recently we have been consulted by a 58-year-old woman who had been apparently "successfully" treated over a period of thirty years. Indeed, she had been skillfully managed, for which she was grateful; yet she plaintively requested surgery at this late date, for while she had lived, "It wasn't like a life. I never could do anything."

Since medical therapy has so little to offer in many cases, and since a stenotic lesion is essentially a mechanical stricture, it is little wonder that direct surgical attack was suggested by Brunton as early as 1902.² The ideal approach to such a problem can be directed only at correction of the stenotic valve itself, provided this can be accomplished without the production of deleterious side effects. Should such attempts fail, satisfactory relief might be forthcoming by less definitive but fairly adequate indirect methods. When a condition is recognized as offering only a fatal or hopeless outlook, desperate measures seem less desperate and with application and courage not infrequently can be made safe.

From the Thoracic Surgical Services of the Episcopal Hospital and the Hahnemann Medical College and Hospital, Philadelphia, Pa., and The Memorial Hospital, Wilmington, Del.

Read at the Twenty-ninth Annual Meeting of The American Association for Thoracic Surgery, New Orleans, La., March 29-31, 1949.

ANATOMIC, PATHOLOGIC, AND PHYSIOLOGIC CONSIDERATIONS

The interplay of these three factors with reference to the mitral valve may seem complicated, but when reduced to essential components is readily understandable. One may think of the normal mitral valve as a truncated cone or megaphone composed of thin, flexible membrane. Its apical portion is suspended by many guy wires (chordae tendineae) which are grouped more heavily in two areas—one on each side of the apex of the cone. These areas represent the anatomic fusion of the two components of the valve (an anteromedial and a posterolateral leaf), and when modified by rheumatic disease become the “commissures.” The entire cone is somewhat flattened toward the apex and these “commissures” represent the lines of folding (lateral seams of the megaphone) of the valve structure. The plane of flattening is neither horizontal nor vertical as the patient lies supine, but is rather sharply oblique. The anteromedial cusp is larger than the posterolateral, lies diagonally anterior to it, and is continuous with the posterior wall of the intracardiac portion of the aorta. In auricular systole the posterolateral cusp lies well away from the lateral ventricular wall, but during ventricular systole may approximate it. For this reason, mitral regurgitation is primarily a dysfunction or defect of the anteromedial cusp of the valve. Contraction of the papillary muscles synchronous with ventricular contraction draws the chordae tendineae taut and prevents the valve leaflets from becoming inverted or displaced backward into the auricle.

In rheumatic disease the mitral valve develops numerous minute cauliflower-like vegetations (1 to 2 mm. diameter) in a row along the line of closure of the valve. Healing leads to the formation of scar tissue. With repeated infection and healing there is gradual development of fibrosis, thickening, narrowing, and shortening of the apical one- to three-quarters of the mitral cone. The apical end becomes fixed in the infolded or flattened position, so that the “commissures” become an anatomic reality. Since the fibrosis and fixation of the orifice take place during constant heart motion, the orifice assumes a slightly oval shape. Thus, in a well-developed stenotic lesion the valve is reduced to a rigid slit in a fibrotic plaque. There is then marked resistance to the passage of blood from the left auricle into the left ventricle. There is also, inevitably, some degree of regurgitation of blood into the left auricle through the rigid slit during ventricular systole.

When this regurgitation is minimal, such a lesion is considered to be one of “pure” mitral stenosis. Such a “pure” lesion markedly interferes with left ventricular filling and thus with systemic cardiac output. Many such hearts are unable to increase their output over and above the resting bodily requirements. It is easy to understand why such hearts cannot perform any appreciable amount of work, and why patients with such hearts become dizzy and faint upon exertion.

Concurrently, since the egress of blood from the left auricle is impaired, increased pressure within and great dilatation of this chamber results. The high intra-auricular pressure is transmitted to the entire pulmonary vascular system and thence to the right ventricle. A chronic pulmonary hypertension ensues, with eventual rupture of pulmonary capillaries (hemoptysis), pulmonary edema (dyspnea), and right heart failure (enlarged liver, ascites, and peripheral edema).

SURGICAL CONSIDERATIONS

The possible surgical approaches to the problem of mitral stenosis seem to take one of the following three courses:

1. Methods of by-passing the stenotic mitral valve
2. Methods of relieving the associated pulmonary hypertension
3. Methods of direct surgical attack upon the stenotic valve

Methods of By-passing the Stenotic Mitral Valve.—To our knowledge very little has been attempted or accomplished along the lines of by-passing the mitral valve. Jeger³ suggested in 1913 that a valved vein might be grafted to serve as an anastomosis between the pulmonary vein and the left ventricle, and thereby adequately sidetrack the stenotic mitral valve. Litwak,⁴ working in our laboratory, has been able to produce such a by-pass by anastomosing a pulmonary vein directly to the left ventricle, utilizing a free graft of azygos, hemiazygos, or femoral vein. This was accomplished in five dogs with function observed for several days, but all became thrombosed within two weeks to one month (Fig. 1).



Fig. 1.—Azygos vein graft from inferior pulmonary vein (below) to the lateral wall of the left ventricle (above) produced in the experimental animal.

Satinsky,⁵ also in our laboratory, attempted quite a different type of by-pass. He divided the subclavian artery in dogs and anastomosed the distal end of this vessel to a pulmonary vein. Similar anastomoses of the pulmonary vein to the splenic artery and aorta were also attempted. These operations were deleterious to normal dogs, causing death, but conceivably might be of some value were the conditions of clinical mitral stenosis present.

Methods of Relieving the Associated Pulmonary Hypertension.—Various methods have been devised to relieve the associated pulmonary hypertension which accompanies mitral stenosis:

(1) Harken* has suggested that a measure of improvement may be afforded by removal of the cardiac accelerator and afferent nerves to the heart. Relief so obtained is through the production of a slower heart rate, thereby increasing ventricular filling time, or by the interruption of pain fibers. He has observed symptomatic relief in a patient so treated, but suggests that such an approach can obviously only be one of palliation. We have had no experience with this form of treatment.

(2) Attempts have been made to destroy the function of the tricuspid valve with the production of tricuspid regurgitation to prevent easy access of blood into the right ventricle and lower its pulmonary output.⁷ In our opinion such an approach has little to recommend it.

(3) The production of a communicating shunt between the pulmonary and systemic venous systems, thus affording a measure of relief to the hypertension within the left auricle, has some merit. An anastomosis between the azygos and pulmonary veins has been accomplished by Sweet* with subjective relief of distressing pulmonary symptoms.

(4) Both Harken* and our group have produced interauricular septal defects to relieve the pulmonary hypertension of mitral stenosis. Such a venous shunt will reduce the hypertension and strain within the left auricle and, secondarily, the associated hypertension throughout the pulmonary vascular bed. Bing¹⁰ and our laboratory have obtained pressure readings within the pulmonary artery in certain cases of mitral stenosis which were higher than that within the aorta (120+ mm. Hg). Theoretically, a venous shunt should relieve strain on the entire lesser circulation, and one would anticipate relief of such symptoms as hemoptysis and acute attacks of pulmonary edema. It is probable that such an effect is accomplished to a degree.

Unfortunately, reduction of the left intra-auricular pressure without enlargement of the mitral orifice results in a destruction of the compensatory mechanism which nature has set up to force blood through the narrowed mitral valve. With the production of such a shunt, the left ventricular output falls. The amount of fall is related to the relative sizes of the shunt orifice and the mitral orifice. Obviously, no case can withstand a shunt if the cardiac output during exercise does not materially increase over the resting requirements. By the same token, it is essential even in those who can so increase their output, that this should not be cut below the level of resting requirements. Since most clinical cases can do no more than double their resting output, it follows that one should not produce an opening even as large as the stenotic mitral orifice unless it is planned to render the patient totally bedfast.

It has been repeatedly stated that Nature, herself, has produced a similar combination of defects in the form of Lutembacher's syndrome. Indeed, the syndrome does embrace both a mitral stenosis and a large auricular septal defect. In this instance it must be remembered that compensation has been established

over many years by gradual changes in the heart and by a great increase in the total blood volume. These patients are said to do well, or at least somewhat better than do cases of "pure" mitral stenosis. Taussig,¹¹ with reference to this syndrome, stated that, "The blood so shunted [through the auricular septal defect] passes into the right ventricle and thence is pumped out through the pulmonary artery to the lungs and is again returned by the pulmonary veins to the left auricle. Thus an excessive amount of blood is pumped around and around the lesser circulation; whereas the left ventricle, aorta, and systemic circulation receive less than their normal quota of blood. The right auricle and ventricle are enlarged. The pulmonary artery is usually twice the size of the aorta. The strain on the left auricle is relieved by the defect in the auricular septum. Therefore, the left auricle is not enlarged. The left ventricle is small." Thus, for a time the strain on the left auricle and pulmonary vascular bed may be somewhat relieved, but only at the expense of producing a similar strain on the right ventricle, a condition equally serious. Again quoting Taussig,¹¹ "The



Fig. 2.—Lutembacher syndrome showing enormous dilatation of the pulmonary artery. Anteroposterior and right anterior-oblique positions. (From Taussig: *Congenital Malformations of the Heart*, Commonwealth Fund.)

late development of cardiac difficulties [in Lutembacher's syndrome] occasionally occurs after a relatively minor illness. For example, a patient who has never been known to have any cardiac abnormality, after some *slight* illness may suddenly develop symptoms which lead to progressive heart failure." (See Figs. 2 and 3.) It would seem that any condition which caused such great pulmonary arterial and right ventricular enlargement can scarcely be considered desirable. Actually this defect terminates fatally at the average age of 40 years—slight, if any, improvement over longevity in uncomplicated mitral stenosis.

Indeed, the production of a venous shunt for mitral stenosis seems somewhat comparable to the production of an arterial shunt (systemic artery to pulmonary artery) for the treatment of congenital pulmonary stenosis. The production of an artificial ductus arteriosus is lifesaving in cases of severe pulmonary stenosis. However, too large a shunt is promptly fatal, and any arterial shunt increases the load on the myocardium, leading to eventual cardiac enlargement. Helpful as these shunts have been, if a method of direct and successful attack upon the

pulmonary stenosis were to become available, we have no doubt that all concerned would promptly embrace the more direct procedure. It would then be necessary, although perhaps not practicable, to recall these cases for the performance of direct valvular surgery and subsequent division of the artificially produced "patent ductus arteriosus." So, too, with the venous shunt. If such shunts are created to save life in the face of demonstrably superior direct valvular attack, we will soon be faced with the problem of repairing these shunts.

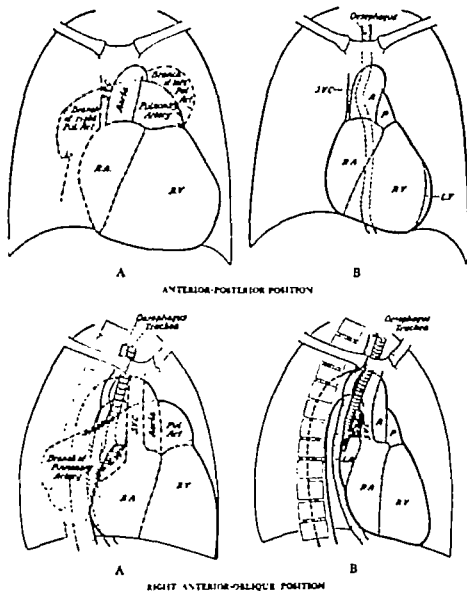


Fig. 3.—(A) Lutembacher syndrome with dilated pulmonary artery and (B) normal heart, adult. (From Tausig: Congenital Malformations of the Heart, Commonwealth Fund.)

Methods of Direct Surgical Attack Upon the Stenotic Valve.—Direct attack upon the mitral valve undoubtedly has been contemplated for many decades. It was proposed by Sir Lauder Brunton² in 1902. Being a physician and not a surgeon, he had to be content with suggesting that the only proper and logical approach to the problem of mitral stenosis was to "lengthen the slit." How sound his judgment was will become evident shortly.

Since the first section of the mitral ring by Cutler¹² in 1923, there have been a number of direct attacks upon the stenotic mitral valve. The approach and the methods employed have varied. Thus, the left auricular appendage as a site of entrance has been utilized by Allen and Graham, 1922¹³; Souttar, 1925¹⁴; the authors, 1945; Smithy, 1948¹⁵; and Harken, 1948⁶; the left ventricle by Cutler, Levine, and Beck, 1924¹⁶; Pribram, 1926¹⁷; Smithy, 1948¹⁵; and through the left

pulmonary vein by Harken, 1948.⁶ It is our considered opinion that the approach through the auricular appendage is far superior to any other. It leads directly into the wide opening of the mitral funnel and thence to its stenotic orifice. There are no chordae tendineae to interfere with the passage of the instrument or finger. In addition, no serious disturbance of cardiac rhythm or function is produced. The finger or instrument is well tolerated in the roomy left auricle, unless the actual passage of blood into the ventricle is obstructed for more than three beats. In addition, the appendage may be readily and securely ligated at completion of the operation.

On the other hand, the left ventricular approach is obstructed by chordae tendineae and does not insure ready or accurate localization of the small opening of the mitral funnel. Such an approach may provoke a serious arrhythmia, does not permit digital insertion or palpation, and may be difficult to close securely.

Approach through a pulmonary vein is less desirable than the auricular route, but may be an improvement over the ventricular method provided the vein is not so damaged as to require ligation.

Methods of dealing surgically with the valve have included (1) simple incision of a valve cusp, (2) excision of a portion of the mitral ring, (3) digital dilatation of the stenotic orifice, (4) valvuloplasty, and (5) commissurotomy.

Simple incision of a mitral valve cusp has a very deleterious effect upon the experimental animal. If the anteromedial or "aortic" cusp of the mitral valve is completely divided, death is prompt and almost immediate. If the posterior cusp is completely divided, death is usual within twenty-four hours. While Cutler's first patient lived four and one-half years after simple incision, it appears that he did not completely divide the cusp. Dogs, too, will tolerate lesser degrees of valve section. One of the objections to simple incision of the ring is the possibility of healing at the site of the incision. It seems improbable that such healing would occur if the valve ring were widely incised, since the edges would then gape widely during most of the cardiac cycle. On the other hand, if the incision extended only partially through the scar tissue, it could not gape and very probably healing and further cicatrix would occur. Of three such cases reported by Cutler, Levine, and Beck,¹⁰ two died shortly after surgery. Contained in this report are two additional cases, both of whom died from severe regurgitation (Cases 8 and 9).

Simple excision of the valve ring was practiced by Cutler and Levine, 1923¹⁸; and Cutler, Levine, and Beck¹⁰; also by Pribram, and later by Smithy. Since it was felt that the only hope in mitral stenosis was to replace it by a regurgitant type of lesion, partial excision of the valve orifice seemed to be a logical procedure. We are unequivocally opposed to this concept, since experimentally and clinically it has been demonstrated that a suddenly produced mitral regurgitation is poorly tolerated and is as serious a lesion as the original stenosis. Bitter experience in two of the cases presented makes us adamant on this point (Cases 8 and 9).

Results by this form of treatment have been discouraging in the past. Thus, four patients so treated by Cutler, Levine, and Beck¹⁰ and one by Pribram, all died within six days after operation. Smithy reported five living patients out of seven such operations in June, 1948. His better results may be attributed partially to advances in anesthesia and surgery since the work of the 1920's. More pertinent, however, is the fact that by design he excised relatively small pieces of valve tissue. Time will supply the answer as to how well his patients will carry on with their admitted regurgitation.

Digital dilatation of the stenotic valve was first practiced by Souttar in 1925 with success in one case. We have since performed three such dilatations with one success. The first of these was performed on June 12, 1946. A very hard, calcified valve slit in a practically moribund woman was dilated with marked temporary improvement. Death within three days was disclosed at autopsy to be due to clotting at the torn commissures. It was evident that the valve had torn at the line of the commissures sufficiently to establish some temporary valve function. Thrombosis had quickly re-established or increased the stenosis, causing death. At post-mortem examination the idea of cutting the commissures well into normal valvular tissue under direct digital guidance or "vision" was born (Case 2). Since that time we have had to resort to simple digital dilatation in two additional cases, with one success (Cases 12 and 13). We are well aware from studies upon stenotic mitral valves incidentally found at autopsy that simple digital dilatation does not always result in tearing the fused fibrotic commissures. The ring tears at its weakest point, which may well be across a cusp. Even when the tearing does occur at the commissures, one finger is seldom large enough to force the tearing to extend beyond the fibrotic tissue into normal valve structure. However, this early case of dilatation did result in what we then considered to be an entirely new concept of treatment. We now believe that Sir Lauder Brunton had much the same idea.

Harken¹¹ used the term *valvuloplasty* to describe his procedure of resection of portions of the valve ring at the commissures. He is still of the opinion that relief of stenosis is by the production of regurgitation. He rightly recognized that resection will best be tolerated if performed at the commissures, assuming that resection with regurgitation is the desired result. Two such operations have been performed. In one the pressure in the left auricle was 450 mm. water, but rose above the readings of a 500 mm. manometer after valvuloplasty of this type. This was attributed to a coincident tachycardia. The patient died in pulmonary edema twenty-four hours later. In the other patient, left auricular pressure dropped from 450 mm. water to 400 mm. during the procedure. He was improved subjectively. Localization of the commissures was accomplished by palpating the valve with the valvulotome inserted through the auricular appendage.

Commissurotomy is a term suggested to us by Dr. Thomas Durant of Philadelphia to designate the procedure which we have employed to re-establish a marked degree of normal mitral valve function.¹² Our present operative technique is simplified and direct. With the patient in the supine position, the left arm over the head, an incision is made over the left third rib or in the infra-

mammary fold. The third rib is removed, including the costal cartilage. The cartilages of ribs 2 and 4 may be divided. The pleura is opened through the incised rib bed. The lung is permitted to collapse. The pericardium is incised longitudinally, one-half inch anterior to the phrenic nerve, for three inches. Care must be taken not to extend the incision too far toward the apex, lest the heart have a tendency to herniate from its sac. The huge distended auricular appendage then protrudes (Fig. 4). After spraying 2 per cent procaine into the pericardial sac, it is injected under the epicardium, encircling the base of the auricular appendage (at the suggestion of Smithy). A purse-string suture of heavy braided silk on an atraumatic needle is passed about the appendage through this injected area. A Satinsky clamp²⁰ is then closed over the base of the appendage and a generous portion of its tip is amputated (Fig. 5). This piece serves for heart muscle biopsy. All intra-appendageal trabeculae that might interfere with insertion of the finger are divided. Two gloves are worn on the right hand. An opening is made in the outer one on the palmar surface at the base of the index finger, and another at the tip. The blade of the commissurotomy knife is inserted between the gloves (Fig. 6), on the index finger, and the finger is inserted into the left auricle as the clamp is released (Fig. 7). The finger is well tolerated by the auricle and causes no disturbance unless the mitral opening is actually obstructed by the finger tip for several beats. The valve is easily located, and is explored. Its structure is estimated, and the size of the opening and the location of the commissures are determined. The knife is then protruded through the orifice and the hook is engaged upon the anterolateral commissure (Figs. 8 and 9). A backward stroke divides the commissure, usually adequately the first time. The finger again palpates the opening and gently dilates it. Sometimes there are remaining fibrous strands which require either digital separation or actual division with the knife. If the cut in the commissure does not extend well into normal tissue it is repeated. In cases which have a rather soft valve orifice markedly diminished in cross-sectional area, simple opening of the lateral commissure is, in our opinion, sufficient. On the other hand, with a rigid, fixed, and sometimes calcified valve, incision of only one commissure would undoubtedly be inadequate.

We are indebted to Harken for a practical point brought to our attention recently with regard to the execution of our next maneuver. Prior to this suggestion, we had been using a metal sheath on the finger to permit changing from a left cutting to a right cutting knife and thus facilitate the division of both commissures. It was necessary by this former method markedly to supinate the wrist to incise properly the posteromedial commissure, and even then we had failed to cut this commissure properly in two cases. As a result, both of these patients died of severe regurgitation because the incision had been carried across the cusp slightly anterior to the medial commissure and into the so-called "aortic baffle"—the wall of the intraventricular portion of the aorta. One of these patients died within twenty-four hours, the other within three months. Autopsy disclosed the condition in both patients. There was no sign of healing of the incision in the longer surviving patient. Harken pointed out that by simply pronating the wrist the left cutting knife could be used for both commissures and



Fig. 4.



Fig. 5.

Fig. 4.—Dilated auricular appendage protruding through incised pericardium.

Fig. 5.—Purse-string suture has been placed about the base of auricular appendage and temporary clamp applied.



Fig. 6.—Commissurotomy knife inserted between two gloves on the palmar surface of the right index finger.

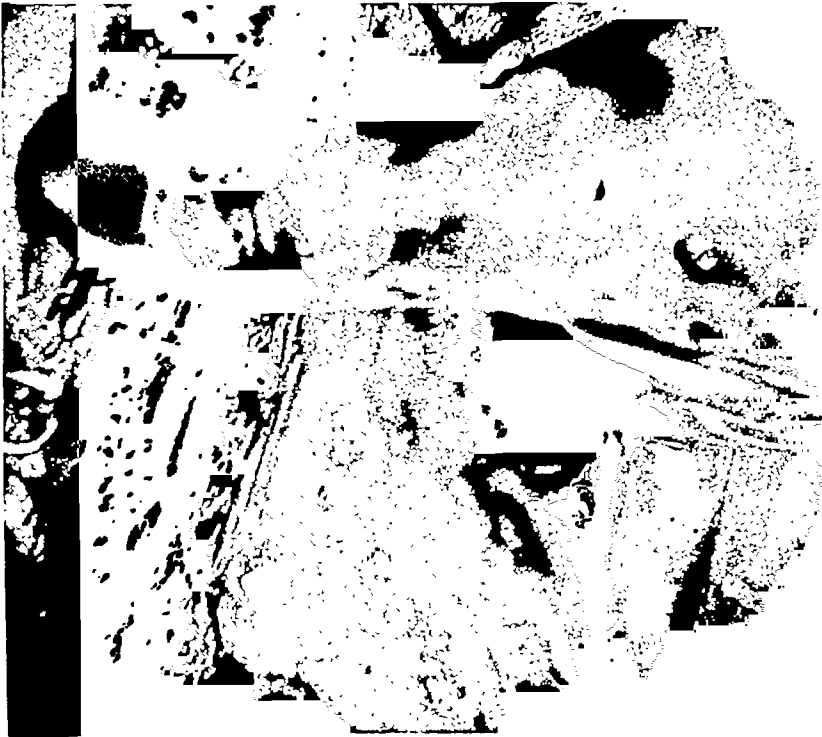


Fig. 7.—Right index finger and commissurotomy knife inserted through opening in left auricular appendage. Note handle of knife at upper center.

the cut on the medial side could be directed more posteriorly. Such a technique removes the necessity for the sheath with its extra bulk and the increased regurgitant blood loss which occurs between knife and sheath.

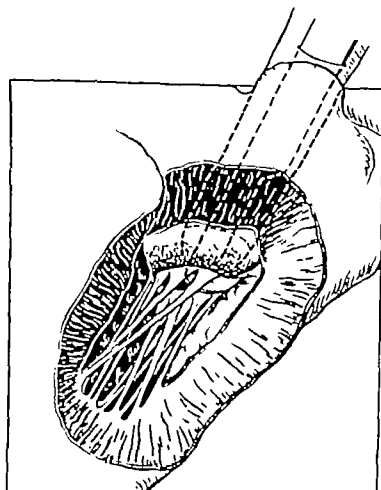


Fig. 8.—Tip of index finger at stenotic mitral orifice with hooked knife blade applied to anterolateral commissure.

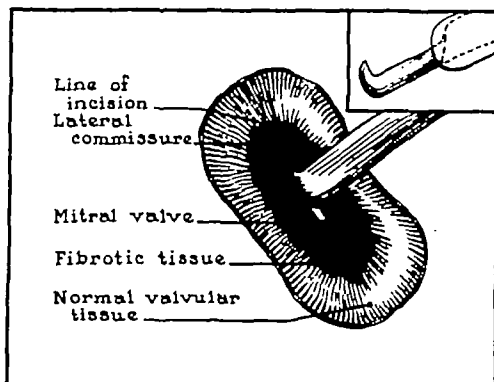


Fig. 9.—Diagrammatic representation depicting position of commissurotomy knife incising anterolateral commissure well into normal valve tissue.

We now, therefore, reinsert the knife through the orifice by simply pronating the hand, bringing the blade into apposition with the posteromedial commissure, which is then divided. In order to handle cases with a very hard or

calcified valve, we have prepared a special narrow-bladed backward-cutting punch (Fig. 10). This has a long bite so that it, too, will cut completely through the diseased tissue and into the flexible membranous valve. Thus we still obtain practically the same effect as by simple incision of the commissures but without the advantage of digital guidance. It is sometimes difficult accurately to delineate the commissures even by digital palpation. Blind palpation with an instrument inserted through the cardiac wall seems dreadfully inaccurate and inadequate. Perhaps some method of direct vision within the heart would be more advantageous. Allen and Graham²¹ have devised a cardioscope with which the valve can be visualized with some accuracy. We have not had the advantage of this instrument. Until such an instrument becomes readily available and is of proved value, simple digital "vision" seems practical and adequate.

Having inserted the finger once more through the enlarged opening to make sure of its mobility and function, the finger and knife are deftly withdrawn from the auricle. The previously placed purse-string suture is drawn tight, preventing more than a few cubic centimeters blood loss. The suture is tied, and the cut edge of the appendage is oversewn (Fig. 11). There should be no disturbance of the systemic circulation and no irregularities throughout the procedure. The pericardium is left open and the pleura is drained. The chest wall is closed in layers.

Postoperatively, the patient may experience considerable pain for three to four days from the "chemical" pericarditis produced by manipulation. This is controlled by opiates. Ambulation is permitted within two to five days.

A word concerning the anesthetic methods employed may be in order at this point. The suggestions of Dr. Kenneth K. Keown, worked out in the departments of Dr. Frank Sena at the Episcopal Hospital and Dr. Henry S. Ruth at Hahnemann Hospital, have been followed. Preoperative medication has consisted of nembutal or Seconal, gr. $1\frac{1}{2}$, sixty to ninety minutes prior to the scheduled surgery; demerol, 75 to 150 mg., given thirty minutes later; and atropine sulfate, gr. $1/100$ to $1/200$, thirty minutes before induction. Intravenous procaine HCl in 0.2 per cent solution is begun fifteen minutes before sodium pentothal-curare induction. Oral intubation is carried out with this preparation, and procaine intravenous solution continued throughout the operation at the rate of 1 Gm. per hour for analgesia with additional sodium pentothal for hypnosis. High concentrations of oxygen are employed throughout. Nitrous oxide-oxygen (50%, 50%) for additional analgesia may be necessary. On this regime the patient is usually awake at the completion of surgery.

It is our present belief that commissurotomy by such a technique is a simple, relatively safe, direct method of re-establishing a normal type of valve function. It has the great advantage of direct digital guidance. It should not, and has not, in any of our four successful cases, produce any detectable amount of mitral regurgitation. Blood loss is minimal and no apparent disturbance to heart function results. That it effectively accomplishes the desired result is shown not only by the marked clinical improvement in all living patients, but also by objective physiologic studies. Thus, in a recent case (studied with the aid of

the Lilly electronic manometer) the pressures within the left auricle taken at the time of surgery fell from 45.2/30.3 mm. Hg (610/409 mm. H₂O) to 14.5/2.6 mm. Hg (195/35 mm. H₂O) fifteen minutes later (after commissurotomy). This interval had not been marked by blood loss (30 c.c.), cardiac arrhythmia, nor

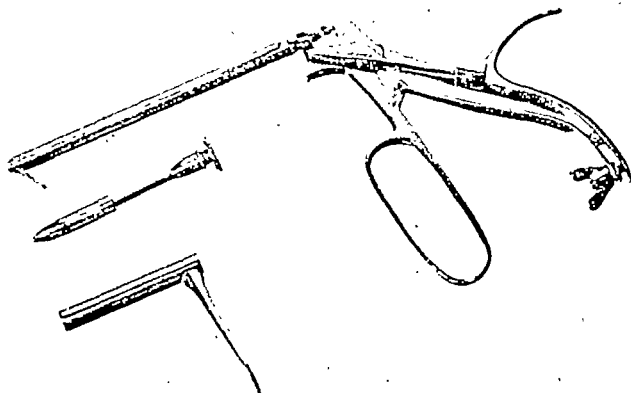


Fig. 10.—Narrow-bladed commissurotomy punch with trocar and cannula.



Fig. 11.—Auricular appendage ligated at base and oversewn at tip after commissurotomy.

calcified valve, we have prepared a special narrow-bladed backward-cutting punch (Fig. 10). This has a long bite so that it, too, will cut completely through the diseased tissue and into the flexible membranous valve. Thus we still obtain practically the same effect as by simple incision of the commissures but without the advantage of digital guidance. It is sometimes difficult accurately to delineate the commissures even by digital palpation. Blind palpation with an instrument inserted through the cardiac wall seems dreadfully inaccurate and inadequate. Perhaps some method of direct vision within the heart would be more advantageous. Allen and Graham²¹ have devised a cardioscope with which the valve can be visualized with some accuracy. We have not had the advantage of this instrument. Until such an instrument becomes readily available and is of proved value, simple digital "vision" seems practical and adequate.

Having inserted the finger once more through the enlarged opening to make sure of its mobility and function, the finger and knife are deftly withdrawn from the auricle. The previously placed purse-string suture is drawn tight, preventing more than a few cubic centimeters blood loss. The suture is tied, and the cut edge of the appendage is oversewn (Fig. 11). There should be no disturbance of the systemic circulation and no irregularities throughout the procedure. The pericardium is left open and the pleura is drained. The chest wall is closed in layers.

Postoperatively, the patient may experience considerable pain for three to four days from the "chemical" pericarditis produced by manipulation. This is controlled by opiates. Ambulation is permitted within two to five days.

A word concerning the anesthetic methods employed may be in order at this point. The suggestions of Dr. Kenneth K. Keown, worked out in the departments of Dr. Frank Sena at the Episcopal Hospital and Dr. Henry S. Ruth at Hahnemann Hospital, have been followed. Preoperative medication has consisted of nembutal or Seconal, gr. $1\frac{1}{2}$, sixty to ninety minutes prior to the scheduled surgery; demerol, 75 to 150 mg., given thirty minutes later; and atropine sulfate, gr. $1/100$ to $1/200$, thirty minutes before induction. Intravenous procaine HCl in 0.2 per cent solution is begun fifteen minutes before sodium pentothal-curare induction. Oral intubation is carried out with this preparation, and procaine intravenous solution continued throughout the operation at the rate of 1 Gm. per hour for analgesia with additional sodium pentothal for hypnosis. High concentrations of oxygen are employed throughout. Nitrous oxide-oxygen (50%, 50%) for additional analgesia may be necessary. On this regime the patient is usually awake at the completion of surgery.

It is our present belief that commissurotomy by such a technique is a simple, relatively safe, direct method of re-establishing a normal type of valve function. It has the great advantage of direct digital guidance. It should not, and has not, in any of our four successful cases, produce any detectable amount of mitral regurgitation. Blood loss is minimal and no apparent disturbance to heart function results. That it effectively accomplishes the desired result is shown not only by the marked clinical improvement in all living patients, but also by objective physiologic studies. Thus, in a recent case (studied with the aid of

fever at the age of 10 years, two previous grand mal seizures, and alcoholism. There were two previous hospital admissions—May 23, 1943, and Aug. 19, 1944—for cardiac decompensation. At this admission he complained of weakness, fatigue, repeated attacks of hemoptysis, and dyspnea, with precordial and epigastric pain. Physical examination revealed marked cardiac enlargement, apex beat in the sixth interspace anterior axillary line, totally irregular pulse rate of 110 with pulse deficit; at the apex, a diastolic murmur ending in a snapping first sound was heard. P_2 and A_2 were increased in intensity. There was auricular fibrillation, no precordial thrill, and blood pressure was 100/64. Dosage of digitalis was readjusted as the patient had not followed his digitalization program faithfully.

Routine laboratory studies were within normal range. Teleroentgenograms revealed marked enlargement to the right, moderate to the left, with marked indentation of the esophagus by the left auricle seen on lateral views. Chest x-ray examination for lung fields revealed chronic thickening of the root shadows but no extension beyond the hilar zone. Electrocardiograms confirmed the clinical finding of auricular fibrillation with myocardial degeneration. Venous pressures were slightly elevated, circulation time increased, especially arm to tongue; exercise tolerance diminished.

On Sept. 24, 1945, intercostal nerve block of the left first to fourth thoracic ganglia was performed with 1 per cent procaine and repeated with absolute alcohol after demonstration of some relief of anterior chest wall pain.

After considerable rest and care designed to improve his general condition, the patient was placed on preoperative penicillin and quinidine (to reduce cardiac irritability). On Nov. 14, 1945, mitral valve exploration was attempted under cyclopropane, oxygen, and ether endotracheal anesthesia. Coincident with the placing of purse-string sutures in the extremely friable auricular appendage, uncontrollable hemorrhage resulted and the patient died on the table despite all attempts to obtain hemostasis.

CASE 2.—W. S., a white woman, aged 29 years, was admitted to Hahnemann Hospital, May 4, 1946, with the diagnosis of rheumatic heart disease with mitral stenosis. There was a history of scarlet fever and acute rheumatic fever at the age of 7 years. Since that time there had been many transient attacks of malaise and joint pains. In 1935, there was an onset of ankle and generalized edema. In 1939, congestive failure again developed during pregnancy. Therapeutic abortions were done in 1941 and 1944. She had been on and off digitalis therapy since 1935. On admission prior to surgery, the patient had a temperature elevation to 101° F., sore throat, and nausea. Rales were present in both lungs, as were edema of thighs, legs, and ankles, with ascites, liver edge felt 7 cm. below costal margin, and marked venous engorgement. The heart was regular in force, rate, and rhythm; pulse 92, blood pressure, 100/80. It was enlarged both to the right and the left; a diastolic murmur was present at the apex, P_2 was accentuated. Teleroentgenograms indicated most marked enlargement of the right heart border and to a lesser extent the left ventricle. The left auricle bulged into the esophagus. Routine laboratory studies were not unusual except for albumin in the urine. Electrocardiographic tracings showed a sinus rhythm with marked right axis deviation, deformed P waves in all leads, T waves flat in lead one, P-R interval .2 seconds.

The patient was given digitalis treatment, diuretics, analgesics, quinidine, vitamin K, and penicillin. There was marked improvement during the ensuing weeks. Immediately prior to surgery examination of the heart revealed a harsh presystolic mitral murmur with a sharp mitral first sound, a soft short systolic blow, P_2 greater than A_2 , rate and rhythm regular. Exercise tolerance had improved but was not normal; circulation times and venous pressures had returned to normal. The patient was operated on June 12, 1946, and the mitral valve dilated manually. Originally a special backward-cutting punch had been inserted, but the mitral orifice could not be located with this instrument. The valve orifice was felt to split at both angles. Immediately following this maneuver the blood pressure rose to 130/80 and her general condition and circulation greatly improved. After reacting from anesthesia a grade 2 systolic blowing murmur was heard with a slight diastolic component. There was no accentuation of P_2 . A biphasic friction rub was heard. Blood

pressure was 114/76, pulse 100, temperature 100.4° F. Improvement continued for forty-eight hours, when the patient went into sudden collapse, no pressure or pulse obtainable. There was marked dyspnea, heart sounds distant, moist râles throughout lungs, liver edge again down to the preoperative level. Therapeutic measures included pleural aspiration of 150 c.c. bloody serum, positive pressure oxygen mask, innumerable cardiac stimulants, but were of no avail. The patient died at 10:45 P.M., June 14, 1946.

Autopsy Findings.—

Heart: No antemortem thrombi in either the pulmonary artery or the left arterial appendage were found. There was dilatation of the right atrium, marked hypertrophy, and dilatation of the right ventricle, the right ventricular wall measuring 7 mm. in thickness; hypertrophy and dilatation of the left atrium, small-sized left ventricle. There was marked stenosis with calcification of the mitral ring, the ring admitting only the tip of one finger. At the commissures there was clotting and thrombosis in small slits which had been made by surgical finger dilatation. The remainder of the heart valves were relatively normal.

It was during this examination that the idea of more accurate incision of the commissures under direct palpation was formulated.

CASE 3.—W. W., a white man, aged 39 years, was admitted for the seventh time to The Memorial Hospital, Wilmington, Del., this time with the diagnosis of rheumatic heart disease with mitral stenosis. His previous admissions had been occasioned by repeated bouts of hemoptysis regarded to be on the basis of bronchiectasis. On these previous occasions he had had a variety of symptoms in addition to hemoptysis, such as fever, malaise, and chest pain, and chest x-ray examination had revealed transient areas of pneumonitis. Repeated bronchograms had shown cylindrical bronchiectasis of the medial basal division of the right lower lobe, which segment had been removed surgically on April 28, 1947. At the age of 14 years he had suffered an attack of acute rheumatic fever.

Examination of the heart at the time of this final admission revealed the presence of a grade 3 systolic murmur with a low-pitched rumbling diastolic murmur heard at the apex. The heart was compensated, electrocardiogram was normal except for sinus tachycardia, chest x-ray views revealed a mitral configuration; all other studies were within normal range.

Operation was performed on March 22, 1948, and was the first attempt at planned incisional commissurotomy in our hands. The right index finger was inserted through the left auricular appendage, and the mitral valve explored. A large fungus-like mass, roughly 1.5 cm. square, arose from the posteromedial commissure. This protruded into the mitral orifice, making it impossible to incise this commissure. The anterolateral commissure was partially cut and dilated by using an ordinary surgical scalpel on the index finger. This instrument was most unsatisfactory and necessitated a sawing motion at the commissure as it slid over the fibrotic angle. A cut of $\frac{3}{8}$ inch was thought to have been made, plus vigorous dilatation of the valve ring. The necessity for a better cutting instrument obviously resulted from this experience and has led to the development of our present knife (Fig. 6).

Postoperatively the patient was heparinized, it being reasoned that this procedure might prevent the clotting or thrombosis which had occurred in Case 2. This proved to be disastrous, for excessive oozing ensued, requiring repeated pleural aspirations in addition to the usual intercostal drainage catheter. In addition, coincident with the administration of heparin, excessive amounts of fluids were given. The combination of an inadequately performed commissurotomy (autopsy finding), heparin, and an overloaded circulation produced heart failure, terminal pulmonary edema, and death six days after surgery.

CASE 4.—J. R., a white man, aged 29 years, was admitted to the Philadelphia General Hospital in May, 1948, with a diagnosis of advanced rheumatic heart disease. Acute rheumatic fever had developed at the age of 12 years. He was apparently well until May, 1947

(diagnosis of mitral stenosis made in the Army in 1943), when he became dyspneic and edematous. Digitalis and mercurial diuretics relieved his distress. There had been four subsequent hospital admissions for cardiac failure despite continuous digitalization. Thoracentesis at time of last admission yielded 1,500 c.c. Examination upon this final admission revealed mild orthopnea without cyanosis, basal râles in both lung fields, and enlargement of the liver to 19 cm. below the costal margin in the midline. There was no ascites or peripheral edema. Cardiac examination indicated generalized enlargement but a regular, although rapid, rate of 104. A grade 3+ systolic and low-pitched rumbling diastolic murmur was heard at the apex. P₂ was greatly accentuated.

Electrocardiographic tracings and cardiac x-ray pictures confirmed the advanced stage of this patient's heart disease. Venous pressures varied from 115 to 340 mm. of water over a three-week period; circulation times showed great delay from arm to tongue. Ballistocardiographic studies were unsatisfactory.

Surgery was attempted on May 21, 1948, although he was an extremely poor candidate. After incision of the pericardium, the slightest touch of the heart surface produced runs of premature contractions. Despite all known forms of medication, his cardiac hyperirritability increased, ventricular fibrillation developed, and death promptly ensued before intracardiac manipulation of the mitral valve could be attempted.

CASE 5.—C. W., a white woman, aged 24 years, was admitted to the Episcopal Hospital on May 13, 1948, with a diagnosis of rheumatic heart disease with mitral stenosis. Acute rheumatic fever had occurred at the age of 7 years, with no recurrences. Her condition was asymptomatic until 1946, although the presence of a heart murmur had been confirmed repeatedly before this time. In 1946 slowly progressive dyspnea developed, with increasingly frequent attacks of hemoptysis. Her family physician made the diagnosis of mitral stenosis. One attack of cardiac decompensation occurred, treated by digitalization and restricted activity. Despite this regime, dyspnea increased, she was unable to perform her housework or care for her child. On admission examination was relatively normal except for her heart, which was markedly enlarged, particularly the left auricle. There was normal sinus rhythm, a loud, rasping diastolic murmur at the apex, pulse 120, blood pressure 110/72. These findings were confirmed by x-ray examination and fluoroscopy of the heart and electrocardiogram. Routine laboratory studies were within normal limits, venous pressure 110 mm. of water, circulation time arm to lung 12 seconds, arm to tongue 20 seconds. She responded normally to the usual exercise tolerance tests. Ballistocardiographic studies indicated a resting cardiac output of 4.38 liters, which could be increased to 9 liters on exercise.

This patient was considered to be by far the best candidate for surgery whom we had seen to date. Preoperatively she was digitalized and quinidine was given. On June 10, 1948, she was subjected to commissurotomy, which proceeded according to the technique described in this paper without flaw. The anteromedial cusp only was incised. Postoperatively she improved rapidly and was discharged from the hospital on the seventh postoperative day. She returned six weeks later for repeat heart x-ray views and ballistocardiographic studies. The x-ray views revealed considerable reduction in the heart size, particularly of the left auricle. Ballistocardiographic tracings indicated the same resting cardiac output as was present preoperatively, but after exercise this was increased to 12.6 liters. Clinically her improvement, however, was much more marked than these objective findings. In the nine months since surgery she has assumed the entire care of her household and is living a perfectly normal life. Digitalis is now unnecessary.

CASE 6.—S. S., a white woman, aged 35 years, was admitted to the Episcopal Hospital in June, 1948, with a diagnosis of rheumatic heart disease. There was no history of rheumatic fever having occurred during childhood. Indeed, she was well until 1940, when an obstetrician made the diagnosis of a serious heart murmur during pregnancy. During the twelfth week of this pregnancy she developed signs of cardiac failure (cough, pulmonary edema, and shortness of breath), requiring confinement to bed until completion of

gestation. She was then reasonably well until 1946, when she experienced a splenic infarct. In September, 1947, a second splenic infarct occurred, followed in two weeks by a cerebral embolus, causing paralysis of the left side. Motor power completely returned, but she was stricken with a psychosis resembling catatonia. Since that time she has been emotionally unstable, and her speech is slurred. In April, 1948, there was an embolus lodged in the dorsalis pedis artery of the left foot. On admission, in addition to the residual findings of the previously mentioned vascular accidents, her heart was irregular in rate, rhythm, and force—apparently from auricular fibrillation. There was enlargement to the left and a short, rumbling diastolic murmur could be heard at the apex. Blood pressure was 110/80. There were no signs of cardiac decompensation.

Routine laboratory studies were within normal range. Electrocardiographic tracings indicated an auricular rate of 375 and a ventricular rate of 60, interpreted as auricular flutter and flutter fibrillation. Venous pressure, right arm, was 178 mm. water. Circulation time, arm to lung, 9 seconds; arm to tongue, 18 seconds. Fluoroscopy and cardiac x-ray views confirmed the impression of mitral stenosis with enlargement of the left auricle.

The patient was digitalized and penicillin was administered. Immediately before surgery she was given quinidine, gr. 3, every three hours.

Commissurotomy was performed on June 27, 1948. Both commissures were cut using the left- and right-cutting knives inserted through a metal sheath strapped to the index finger, as described in the text. The valve orifice was small and the valve structure similar to muslin. At the conclusion of the procedure the auricular appendage was ligated at its base to prevent further embolic phenomenon.

Postoperative recovery was smooth and without incident. There has been marked clinical improvement, definite diminution in the size of the heart shown by fluoroscopy and cardiac x-ray view. Ballistocardiographic estimations of cardiac output at rest and exercise taken pre- and postoperatively indicate considerable objective improvement.

CASE 7.—F. G., a white woman, aged 35 years, was admitted to the Episcopal Hospital on July 1, 1948, with a diagnosis of rheumatic heart disease. Although there was no definite attack of acute rheumatic fever during childhood, she had repeated bouts throughout her life of epistaxis, and aching in the elbows, ankles, and wrists. She stated that she had always been short of breath. In 1925, during pregnancy, the presence of a heart murmur was discovered. Shortly thereafter, severe hemoptysis occurred and she was bedridden for five months. She underwent cesarean section at term. In 1927 she became pregnant for the second time with a return of the symptoms, at which time she underwent hysterectomy. In 1945 she was hospitalized for cardiac failure. Since that time she has had precordial pain, burning with radiation down both arms, and slight ankle edema.

On admission the patient was mildly dyspneic at rest, and could not walk the length of the corridor without severe dyspnea. She was taking digitalis, gr. 3 daily. Electrocardiographic tracings indicated a ventricular rate of 80 with auricular fibrillation. Cardiac examination showed cardiac enlargement to the left and a diastolic murmur over the apex. Lungs were clear; there was no enlargement of the liver nor peripheral edema. Venous pressure, right arm, was 120 mm. water; circulation time, arm to tongue, 27 seconds. Blood pressure was 120/80. Cardiac x-ray views were compatible with the diagnosis of mitral stenosis.

Commissurotomy was performed on July 13, 1948, in the classical manner. Exploration of the mitral valve revealed the mitral orifice to be too small to permit the insertion of the finger tip. It felt like a buttonhole in a piece of kidskin. The anterolateral commissure was satisfactorily incised and the thrill which was very prominent in the region of the mitral valve disappeared. It was deemed unnecessary to cut the posteromedial commissure. Transfixion sutures of medium cotton are placed through the tip of the auricular appendage and hemostasis was well established. The patient did exceedingly well postoperatively, and was able to traverse the halls with considerably more facility than she could preoperatively, as early as the sixth postoperative day. On the eighth postoperative day, while sitting up in bed talking to her husband, she died suddenly.

Autopsy Findings.—

Heart: A small antemortem thrombus was found in the stump of the amputated (partially) left auricular appendage. The sutures used for this closure were not exposed within the left atrium. The mitral valve appeared patent and the incision in the commissure showed no signs of fibrosis.

Brain: There was generalized edema of the brain, although the vessels did not appear markedly congested. There was a cone-shaped impression on the medulla and the base of the cerebellum, indicating herniation through the foramen magnum. On section of the cerebellum, there was a very definite area of softening, about 2 to 3 cm. in diameter, in the midportion. No obvious hemorrhage in this area was seen.

CASE 8.—H. B., a white man, aged 35 years, was admitted to The Memorial Hospital, Wilmington, Del., on July 22, 1948, with a diagnosis of rheumatic heart disease. There was a history of acute rheumatic fever in 1942. Since that time he has been admitted to the hospital on six occasions prior to the present admission for symptoms of increasing shortness of breath, hemoptysis, and edema. At the time of the present admission, although he was taking digitalis routinely his complaints were that of cardiac decompensation. Physical examination at this time revealed a heart enlarged to the left, normal sinus rhythm, no palpable thrill, marked crescendo, presystolic murmur heard at the apex. There was no sign of pulmonary or peripheral edema; venous pressures were normal as were the circulation times. Cardiac x-ray views were compatible with the diagnosis of mitral stenosis. After a period of care during which the patient received parenteral penicillin, an increased dosage of digitalis, and the institution of quinidine, he was referred for mitral valve surgery.

On Aug. 2, 1948, mitral commissurotomy was attempted. Exploration of the valve revealed the orifice to be very tiny, almost pinhole in type. Because of the marked constriction, no actual commissures could be located. Therefore, the anterolateral cusp was incised in the area assumed to be the general location of the anterolateral commissure. The opening so produced seemed very adequate, so that no further incision was attempted. Postoperatively the patient improved slowly, and there was evidence of a small amount of pulmonary edema. Electrocardiographic tracings showed that a definite change had occurred, in that there was more depression of the S-T segments in the chest leads with low voltage of the T waves in all leads. Clinically the preoperative diastolic murmur was absent and only a slight systolic murmur could be heard. By the ninth postoperative day the patient was out of bed without dyspnea, although fine rales could be heard in both lungs. The patient was discharged in satisfactory condition on Aug. 27, 1948.

He was re-admitted in October, when he was seized with severe chest pain and dyspnea. Physical examination revealed a cyanotic, dyspneic, apprehensive patient, exhibiting pitting edema of both ankles. Cardiac examination indicated that there were two murmurs heard at the apex, one systolic and one diastolic. Although there was some response to mercurial diuretics, there was progressive edema and the patient died in extreme heart failure on Oct. 18, 1948.

Autopsy Findings.—

Heart: The mitral valve was the seat of an operative incision 1 cm. in depth on the lateral aspect of the aortic cusp. There was no evidence of healing of this incision. There were additional calcareous deposits over the surface of the mitral valve.

CASE 9.—J. K., a white man, aged 31 years, was admitted to the Episcopal Hospital on Aug. 20, 1948, with a diagnosis of rheumatic heart disease. He had suffered an attack of acute rheumatic fever at the age of 12 years. After prolonged illness he recovered, and seemed well. In 1940 he passed the Army physical examination and was inducted. In 1943 he experienced repeated episodes of hemoptysis during exercise. At this time he was digitalized, and has remained so to the present admission. History to this time consists essentially of repeated attacks of hemoptysis, increasing shortness of breath, chest pain during paroxysms of coughing, but no peripheral edema. Examination on admission

revealed a patient in no acute distress, but one who became dyspneic while talking. Cardiac examination revealed moderate enlargement, normal sinus rhythm with occasional premature contractions, accentuated P_2 , and a snapping M_1 . There was a rumbling murmur throughout diastole, with presystolic accentuation. There were no signs of congestive failure. Circulation time was, arm to tongue, 19 seconds; arm to lung, 6 seconds. Venous pressure was 160 mm. water. Electrocardiogram revealed a right axis deviation. Cardiac x-ray views were compatible with the diagnosis of mitral stenosis, showing marked left auricular hypertrophy.

Mitral commissurotomy was performed on Sept. 2, 1948, in the classical manner. Mitral exploration revealed a tight fishmouth constriction of the valve orifice. Both lateral and medial commissures were incised. Because of the presence of hard, calcified nodules along the medial commissure, the angle of the medial incision was directed slightly anterior, and a large incision was produced.

The patient responded well immediately after surgery, but during the night began to expectorate frothy, bloody sputum. His course was progressively downward despite all therapeutic endeavor, and he died the next day in acute pulmonary edema.

Autopsy Findings.—

Heart: The mitral leaflets are thickened, rigid, and calcified. The chordae tendineae are shortened and cordlike. There is stenosis of the ring and regurgitation. The latter has been accentuated by surgical incision, which has been carried into the intracardiac portion of the aorta.

CASE 10.—A. W., a white woman, aged 35 years, was admitted to the Episcopal Hospital on Aug. 27, 1948, with a diagnosis of rheumatic heart disease. There was no previous history of rheumatic fever. At the age of 26 years, during her first and only pregnancy, she experienced episodes of marked dyspnea and orthopnea with peripheral edema and precordial pain. The patient's pregnancy was terminated by cesarean section. The subsequent course was that of increasing progressive dyspnea, but no hemoptysis occurred. The patient has been digitalized for several years. Physical examination revealed blood pressure 120/78, pulse 76, cardiac enlargement to the left, P_2 accentuated 2+, diastolic thrill at the apex, rumbling apical diastolic murmur, ending in a snapping M_1 . The lungs were clear, the liver and spleen not palpable.

Preoperative studies included fluoroscopy, cardiac x-ray views, and electrocardiogram which revealed right axis deviation; circulation time with Decholin was 18 seconds, with ether 7 seconds. Venous pressure was 120 mm. water in the right arm. The preoperative preparation included quinidine and penicillin, in addition to the usual care.

Mitral commissurotomy was performed on Sept. 2, 1948. A typical fishmouth fibrotic mitral valve was encountered. Both the lateral and the medial commissures were cut as in the preceding case. Immediately after the medial commissure was incised, the blood pressure dropped markedly and the heartbeat became slow and feeble. This was believed to be due to the loss of an excessive amount of blood through the sheath of the commissurotomy knife inadvertently. The blood was rapidly replaced, with improvement of her condition. The base of the appendage was ligated but no end sutures were placed in the amputated tip of the appendage.

As in the preceding case, the immediate postoperative condition was good, but after twelve hours the blood pressure dropped steadily and despite all therapeutic endeavor the patient died within a twenty-four-hour period. Attempts at cardiac resuscitation by manual massage in bed were to no avail. During this procedure it was noted that considerable hemorrhage had occurred from the stump of the auricular appendage.

This case indicated to us the necessity for both ligation of the base of the appendage and suture of the amputated appendageal tip. These last two cases emphasized to us the undesirability of the sheath which we had been using to facilitate the use of a left- and right-cutting commissurotomy knife. We are grateful to Harken for his suggestions as noted in the text, which have made it possible to dispense with this sheath.

No autopsy was obtained.

CASE 11.—M. R., a white woman, aged 43 years, was admitted to the Episcopal Hospital in September, 1948, with the diagnosis of rheumatic heart disease. There was no history of acute rheumatic fever, but the presence of a heart murmur had been detected in 1930 at the time of examination for life insurance. At the age of 25 years, during her first and only pregnancy, she collapsed from a "heart attack," and the fetus was delivered by cesarean section. At the age of 38 years she experienced a sore throat with fever, which was diagnosed as an attack of rheumatic fever. At the age of 41 years there was cerebral embolization, which affected her speech, the effect lasting for one week. Several months later a second embolus occurred to the right leg, which cleared shortly. In 1947, splenic infarction occurred. The patient had never been in congestive failure, but took .2 mg. digitalis daily for four years. She had taken Dicoumarol, 37.5 mg. daily, since August, 1947.

Physical examination revealed an enlarged heart, auricular fibrillation, marked diastolic mitral murmur, and blood pressure 110/80. There were basal râles in the lung fields, but no other signs of congestive failure.



Fig. 12.—Forceps in interauricular septal defect produced surgically. Note large size compared to stenotic mitral orifice.

Preoperative studies included fluoroscopy and cardiac x-ray examination confirming the diagnosis of mitral stenosis. Venous pressure was 160 mm. water, circulation time, arm to lung, 3 seconds; arm to tongue, 16 seconds. Electrocardiographic studies indicated ventricular rate of 80 with auricular fibrillation and right axis deviation.

Mitral exploration was carried out Sept. 10, 1948. A tiny mitral orifice was found high on the anterior aspect of the mitral valve and was in such a position that commissurotomy could not be performed. Exploration of the interauricular septum was carried out. This revealed a semilunar ridge, somewhat simulating a valve. The commissurotomy knife was engaged upon the tissue proximal to this ridge and an opening was made into the adjacent chamber (right auricle). This opening was then enlarged by two separate diverging strokes of the knife to fashion an interauricular septal defect which would admit

the index finger. Postoperatively the patient responded adequately and remained clear and lucid until death sixty hours later, despite systolic blood pressure ranging from 40 to 60 mm. mercury. All attempts to elevate and sustain a higher blood pressure failed. Oliguria and terminal uremia followed. Our attempts to relieve pulmonary hypertension had so lowered this patient's cardiac output that life could not be maintained. The heart is shown in Fig. 12.

CASE 12.—M. B., a white woman, aged 37 years, was admitted to the Episcopal Hospital on Sept. 7, 1948, with a diagnosis of rheumatic heart disease. She had experienced an attack of acute rheumatic fever at the age of 14 years, with four recurrent attacks after that time. She had been taking digitalis continuously since the first attack. There had been three episodes of marked dyspnea, occurring during pregnancy sixteen years ago, the next in October, 1947, and the third in July, 1948. Edema of the ankles occurred on two occasions, one at the birth of her child sixteen years ago, and again in July, 1948. There was marked exertional dyspnea, but she was able to work with care up to the time of admission.

Physical examination revealed enlarged heart, both to the right and to the left, auricular fibrillation, marked systolic murmur at the apex, with an early diastolic thrill and murmur in the same area. After the usual studies, it was felt that this patient had mitral stenosis and regurgitation of equal severity, and the problem arose as to whether surgery in her case was indicated. Cardiac catheterization studies were unsatisfactory. After consultation with the cardiologist, it was our combined opinion that this patient's course would be rapidly downhill at best, and therefore an attempt at mitral exploration and relief of stenosis was indicated.

Mitral exploration was carried out on Sept. 16, 1948. The mitral valve was found to be very abnormal. The medial half was apparently occupied by a large calcified mass, which was notched at the exact commissure. It was evident that no operation could be performed on this portion of the valve. The lateral half, however, was widely dilated so that the palpating finger could easily be inserted into the left ventricle, even to the apex. The edge of this opening was fibrotic and there was little movement of the valve during cardiac excursion. An attempt was made manually to split and mobilize this stiffened lateral wall, feeling that possibly some motion in the valve might result in improvement. Nothing further was done.

Postoperatively, to our amazement, she has been markedly improved subjectively, although there are no objective findings to corroborate such improvement. There was a marked psychic element in this case which might account for this improvement.

CASE 13.—M. L., a white woman, aged 53 years, was admitted to Doctors Hospital, Philadelphia, on Jan. 16, 1949, with a diagnosis of rheumatic heart disease. Acute rheumatic fever had been sustained three times between the ages of 7 and 10 years. Since the age of 43 years she has had repeated attacks of congestive failure. Physical examination revealed a thin, emaciated woman, prominent neck veins, liver enlarged to 3 cm. below costal margin, moderate ascites, and grade 2 pitting pre-tibial edema. Examination of the heart revealed a loud, blowing diastolic murmur, cardiac enlargement, evidence of auricular fibrillation. There were râles at both bases posteriorly.

Preoperative studies included electrocardiographic tracings indicating auricular flutter; cardiac x-ray views gave evidence of mitral configuration with left auricular and ventricular enlargement confirmed by fluoroscopy. Venous pressure was 120 mm. water; circulation time, arm to lung, 9 seconds; arm to tongue, 27 seconds. Cardiac catheterization was performed and pressure readings in the pulmonary artery were 32.4 systolic and 3.4 diastolic. In the right ventricle pressure readings of 62.3 systolic and 7.6 diastolic were obtained.

It seemed unlikely that surgery was indicated in this case, but at the behest of the patient and the cardiologist who realized the hopelessness of the situation, operative intervention was carried out. Mitral exploration was performed on Feb. 1, 1949. The auricular appendage was found to be tightly adherent to the pericardium and the entire pericardial

space was obliterated by fibrous adhesions. As the auricle and its appendage could not be freed, mattress sutures were placed circularly in the auricular appendage. An incision was made through this area and the finger inserted into the left auricle. The mitral valve was found to be a fibrotic slit somewhat wider anteriorly than posteriorly. The index finger was inserted through the orifice of the valve, which was manually dilated. Tearing at both commissures was accomplished. The finger was withdrawn, and the mattress sutures were drawn tight. This did not suffice to control the bleeding, and a large-sized gelfoam pack was applied over the rent in the auricular appendage. The pericardium was sutured over the area and bleeding was very adequately controlled.

The postoperative period was one of progressive cardiac failure, despite intensive therapy. The patient died within sixty hours.

Autopsy Findings.—

Heart: There was an adhesive pericarditis with multiple calcium deposits, and severe mitral and aortic stenosis, the former being the seat of lacerations at the commissures. Both ventricles were markedly hypertrophied. All other organs were the seat of marked passive congestion.

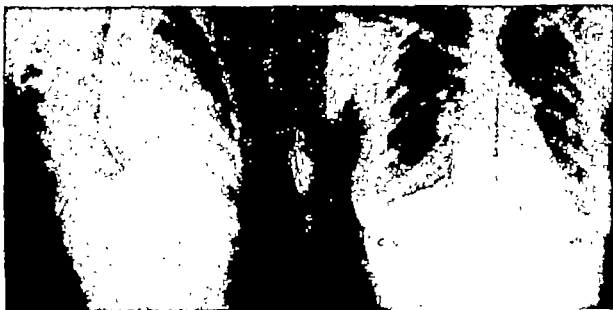


Fig. 13.—Posteroanterior and right anterior oblique roentgenograms showing size of heart and left auricle, preoperative.

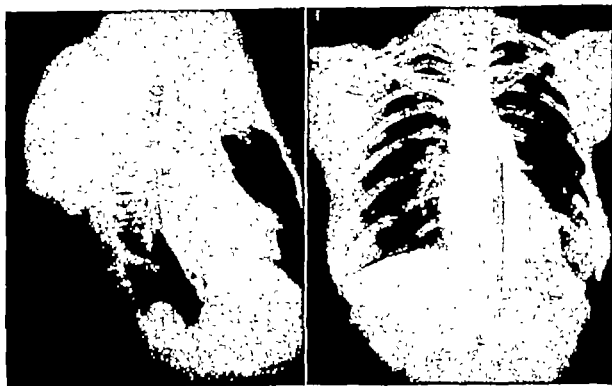


Fig. 14.—Posteroanterior and right anterior oblique roentgenograms showing reduced size of heart and left auricle, postoperative.

CASE 14.—J. B., a white man, aged 32 years, was admitted to the Hahnemann Hospital on Jan. 18, 1949, with the diagnosis of rheumatic heart disease. To the patient's knowledge there was no history of acute rheumatic fever. The patient had noticed increasing dyspnea since 1945, with four episodes of marked hemoptysis. In recent months dyspnea was marked on the slightest exertion. There was no history of congestive failure. Marked cardiac palpitations, but no anginal or substernal pain, were present.

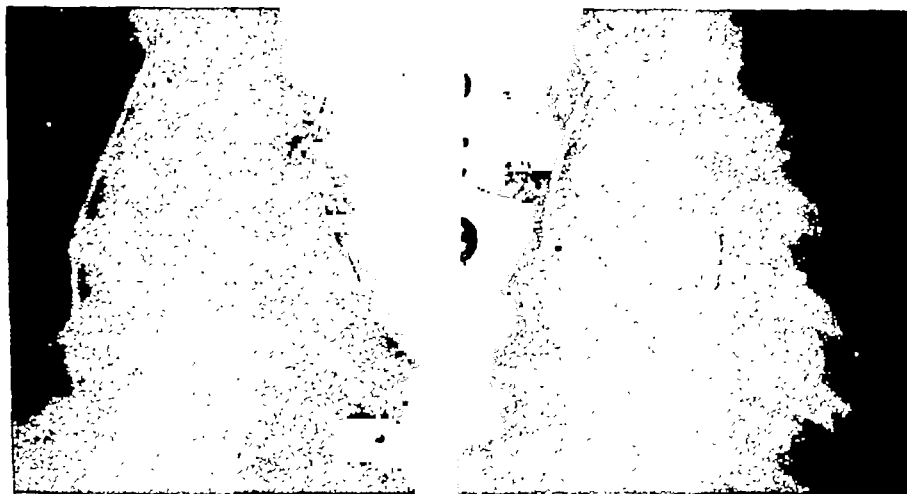


Fig. 15.—Position of catheter in right auricle and ventricle at the time of postoperative pressure studies (Case 14).

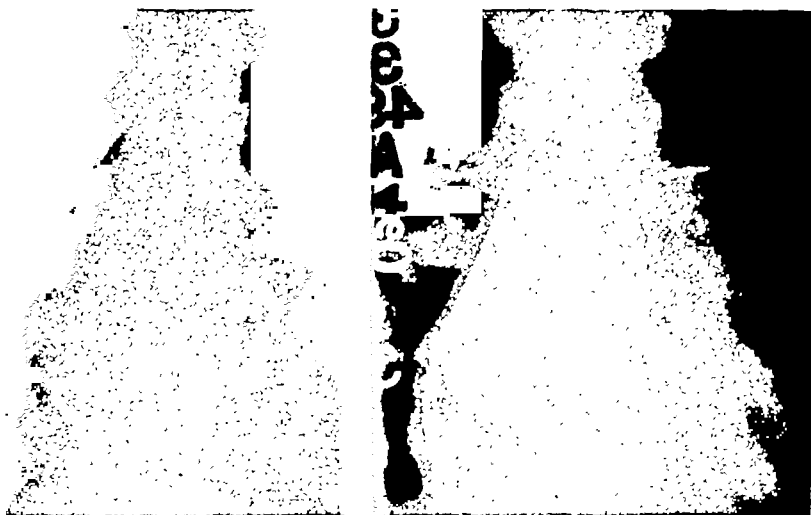


Fig. 16.—Position of catheter in right and left pulmonary arteries at time of postoperative pressure studies (Case 14).

Physical examination of the heart revealed a normal sinus rhythm, early diastolic murmur at the apex, P_2 accentuated, and blood pressure 110/60. Fluoroscopy and teleroentgenograms indicate cardiac enlargement with mitral configuration. Electrocardiographic tracings indicate some auricular dysfunction and myocardial damage with slight right axis deviation. T waves are inverted in Lead CF_2 . P waves are of abnormal duration in Lead 2. Routine laboratory studies were within normal limits. Venous pressure for the right arm was 120 mm. water. Circulation time, arm to lung, was 10 seconds; arm to tongue, 22 seconds. Preoperative cardiac catheterization findings are contained in the text.

Mitral commissurotomy was performed on Feb. 2, 1949, and the mitral slit was found to be so small that it would not admit more than the tip of the finger. The edges of the mitral orifice were folded and beaded. There was a small piece of calcium high on the anterior leaf. The remainder of the mitral valve, both anterior and posterior leafs, was thin and mobile. The lateral commissure was slit by a backward stroke of the commissurotomy knife, and the commissure was divided laterally to the wall of the heart. The opening so produced was large and adequate, and it was felt that medial commissurotomy was unnecessary.

Postoperatively the patient's course was excellent, except for the occurrence of more than the average amount of pericardial pain, which disappeared after one week. The patient was discharged and returned on Feb. 23, 1949, for complete postoperative studies. During this admission, auricular fibrillation came on suddenly, but he reverted to normal rhythm on digitalis and quinidine, which was then discontinued. Venous pressures in the right arm were 95 and 100 mm. water; circulation times, arm to lung, 11 seconds; arm to tongue, 17 seconds. Postoperative catheterization studies (Figs. 15 and 16) as well as pressure readings in the left auricle, before and after commissurotomy, are contained in the text. All pressure readings were recorded on this and other catheterization study cases by a Lilly electronic manometer calibrated in millimeters of Hg. The patient has since returned to work and is now able to carry on the duties (strenuous) of a truck driver in the coal mines.

CASE 15.—S. C., a white man, aged 21 years, was admitted to the Hahnemann Hospital on March 17, 1949, with the diagnosis of rheumatic heart disease. There was no history of acute rheumatic fever, but he was told on routine physical examinations at school and in the Army that he had a "rheumatic heart." He had been repeatedly warned to stay away from strenuous sports, a warning which he had ignored until recently, when he began to notice some slight shortness of breath on exercise. He was a sensitive youth and very heart-conscious. There were no other symptoms to date.

Cardiac examination revealed slight enlargement, normal sinus rhythm, and grade 3 rough diastolic murmur at the apex. There was no associated evidence of any tendency to cardiac decompensation.

Preoperative studies included electrocardiographic tracings which were within normal limits, fluoroscopy, and cardiac x-ray views which revealed very early mitral configuration. Venous pressures in the right arm were 140 mm. water, circulation time, arm to lung, 8 seconds; arm to tongue, 11 seconds. Cardiac output studies determined on the Fick principle indicated a cardiac output of 6.3 liters (cardiac index 3.9) before exercise and 13.5 liters (cardiac index 8.2) after exercise. These figures were not corrected for his preoperative anemia. Complete cardiac catheterization pressure studies were carried out and found to be as follows: Right auricle—maximum systolic 11.9 mm. Hg, maximum diastolic 3.5 mm. Hg; right ventricle—maximum systolic 37.8 mm. Hg, minimum systolic 30.7; maximum diastolic 5.4 mm. Hg, minimum diastolic 0.8 mm. Hg; right pulmonary artery—maximum systolic, 43.5 mm. Hg, minimum systolic 37.0; maximum diastolic 5.2 mm. Hg, minimum diastolic -0.6 mm. Hg. In the main pulmonary artery, maximum systolic, 52.5 mm. Hg, minimum systolic 42.1 mm. Hg; maximum diastolic 4.5 mm. Hg, minimum diastolic -3.4 mm. Hg. The heart rate during these studies varied between 78 and 84 beats per minute. On the basis of these studies, our cardiac consultant stated, "Even though there is no history of rheumatic infection, it is felt that this is a clear-cut case of advanced mitral stenosis. The pulmonic second sound is only 1+ accentuated, and there is no left ventricular enlargement. An exercise tolerance test gave a good response. This case should be a good candidate for commissurotomy, and I see no contraindication to its being done."

Mitral commissurotomy was performed on March 23, 1949, and a valve similar to that described in Case 14 but with a slightly larger orifice was found. Lateral commissurotomy was sufficient to obtain the desired result. During the performance of this operation, pressure readings were obtained before and after commissurotomy from the left auricle. Because of mechanical difficulties in the performance of our electronic manometer,

the readings obtained were unsatisfactory and inaccurate. Objective evidence to corroborate his marked clinical improvement will be obtained in one month from the time of operation, and will be reported at a later date.

Two cases were exploratory or attempted valve operations only, for both patients died before the heart was entered. In the first patient, operated upon Nov. 14, 1945, countertraction on sutures in the left auricular appendage during insertion of a trocar resulted in tearing of the appendage (Case 1). Fatal hemorrhage resulted. The second operation (Case 4) was attempted upon a man in extremely poor condition, who remained cyanotic from the onset of anesthesia. After the pericardium was opened the slightest irritation of the epicardium produced runs of premature contractions. In spite of vigorous intravenous drug therapy designed to reduce cardiac irritability, the heart failed. Drastic resuscitative measures were to no avail.

In two cases a technical error occurred, related to the use of a special metal sheath for the change of knife blades. In both, an incision was inadvertently made across the anteromedial valve cusp rather than through the posterior commissure. Both patients developed severe mitral regurgitation. One died within twenty-four hours and the other within three months (Cases 8 and 9).

In one case an interauricular shunt was produced (Fig. 12). The pulmonary and left auricular hypertension were promptly and adequately relieved. In fact, even the pre-existing auricular fibrillation was abolished. The patient, however, died within three days because she could maintain a systolic blood pressure of only 30 to 60 mm. mercury. By removing the auricular hypertension the compensatory head of pressure necessary to maintain adequate cardiac output had been so lowered that life could not be sustained (Case 11).

In three cases (Cases 2, 12 and 13) simple digital dilatation of the mitral orifice was carried out. Except for the first case (W. S., June, 1946) in which we had no clear-cut ideas of procedure other than to enlarge the stenotic valve, the indication for this procedure rather than commissurotomy was entirely based upon the poor condition of the patient at the time of surgery. In both, the simplest and most rapid procedure seemed advisable. One patient recovered (Case 12) and is, subjectively, remarkably improved. The proper pre- and postoperative set-up for physiologic studies was not available at that time, much to our regret. With our present knowledge none of these cases would have been considered for surgical intervention.

Commissurotomy by the technique described was performed in seven cases with three fatalities. In the first of these latter (Case 3) a modified scalpel was employed, as our present commissurotomy knife had not been perfected. Attempts to incise the commissure were attended by repeated disengagement of the blade as it rode over the fibrotic valve, resulting in an inadequate incision extending only a few millimeters into the scar tissue. Pre- and postoperative pressure determinations in the left auricle were unchanged (320 mm. H₂O). The patient died on the sixth postoperative day in pulmonary edema from unrelieved mitral stenosis. The second patient (Case 7) was operated upon accurately and well, and appeared to be on the way to a most satisfactory result, but suffered a cerebral embolus and died suddenly on the seventh postoperative

day. Autopsy disclosed an adequate and satisfactory commissurotomy. Thrombus formation had occurred in the left auricular appendage, from which the embolus apparently originated. We have observed such thrombus formation many times in animal appendages after valve surgery, but no adverse complications had resulted. We had simply oversewn the incision in the appendage at the conclusion of the procedure. In six succeeding cases (and in one previous, C. W.) we have ligated the auricular appendage at its base to prevent such disaster. The appendage of the third patient operated upon shortly thereafter was ligated at its base without suture of the cut tip. Autopsy indicated that hemorrhage had occurred from this site (Case 10). This experience has led to both ligation at the base and an oversewing suture at the cut tip on all subsequent cases.

The remaining four cases had excellent results. The patients are clinically well. The two patients who had been digitalized for some months prior to surgery no longer need such medication. There is definite diminution in the size of the heart as shown roentgenographically (Figs. 13 and 14). Their tolerance for exercise is remarkably increased, permitting them to return to normal activity. Careful examination has revealed some slight evidence of residual mitral stenosis (by auscultation) in two of the cases (Cases 5 and 14). The others seem to have no residual signs of the disease, although they are admittedly short-term follow-ups. There has been *no evidence of mitral regurgitation* in any of the four.

Comment.—It is essential that these patients be carefully studied and selected for surgery. This should include complete fluoroscopic examination of the heart, posteroanterior and oblique x-ray views including esophagograms, electrocardiographic tracings and phonographic recordings, circulation times, and venous pressures. A careful clinical history and examination are made by an especially interested cardiologist. Catheterization of the right heart is of utmost importance. Pre- and postoperative pressure readings from the pulmonary arteries, right ventricle, and auricle are at present the best objective means of evaluating the results obtained. In certain cases, angiocardiology to determine the size of the left ventricle may be indicated. Ballistocardiograms and cardiac output studies are of great value.

Indications (at present).—

1. Most favorable group:

A. Excessive fatigability.

Increasing exertional dyspnea.

B. No rheumatic activity.

Normal sinus rhythm.

Lesion predominantly stenosis.

Evidence of significantly increased pulmonary hypertension.

2. Less favorable group:

The preceding plus:

A. Recurrent bouts of hemoptysis.

B. Arterial embolic phenomena.

C. Auricular fibrillation without failure.

Hemoptysis in more than amounts necessary to stain the sputum is of grave import. Wolff and Levine²² pointed out that, in their series of cases, the average duration of life following the onset of severe hemoptysis is 32.5 months. Levine²³ stated that the average duration of life following the initial attack of congestive failure is 4.6 years. The development of auricular fibrillation is usually permanent and irreversible. In this state, thrombus formation not infrequently occurs along the endocardium of the dilated and relatively immobile auricular walls. Some 75 per cent of these occur within the lumen of the auricular appendage (left), a common site for the origin of arterial embolization.

Contraindications (at present).—

1. Active rheumatic infection.
2. Presence of superimposed subacute bacterial endocarditis.
3. Cardiac failure uncontrollable by medical means.
4. Presence of marked associated mitral regurgitation or other valve (aortic) deformities.

SUMMARY

Our entire experience with surgery for mitral stenosis—fifteen cases—has been reviewed. In seven of them, commissurotomy was performed with four excellent results and three deaths. Had our present knowledge been applied to all cases, the mortality would have been appreciably lowered.

Commissurotomy is a simple, direct, effective, and safe procedure. Its exact surgical technique has been outlined. It accomplishes relief of mitral stenosis by restoring valve function without the production of additional mitral regurgitation.

Digital guidance in the performance of accurate valve surgery is essential until a method of direct vision becomes established. The left auricular appendage is the most satisfactory avenue of approach to the mitral valve. The auricular appendage must be ligated at the conclusion of the procedure to prevent arterial embolization. This may prove to be the proper approach to the management of arterial embolization in many cases unassociated with mitral stenosis.

An enlarged left ventricle in a case of supposed "pure" mitral stenosis is indicative of some additional significant valve lesion (aortic stenosis or regurgitation or mitral regurgitation) and is probably contraindicative to commissurotomy.

Study methods of valve, in addition to clinical evaluation, embrace cardiac catheterization for determination of pulmonary vascular pressures, ballistocardiography, and other physiologic studies for the determination of cardiac output.

Venous shunts for the treatment of mitral stenosis are of some value, although their eventual effects upon cardiac output, the right ventricle, and the lesser circulation must be kept clearly in mind.

Since this paper was submitted for publication, seven additional mitral commissurotomies have been performed with but one death. Thus of fourteen patients with mitral commissurotomies to date, ten are living and have shown great improvement, both by subjective and objective measurements. Complete data on these additional cases will be presented in a later communication.

REFERENCES

1. White, P. D.: Chronic Valvular Disease, in Textbook of Medicine, ed. 4, edited by Russell L. Cecil, Philadelphia, 1937, W. B. Saunders Company, pp. 1107-1125.
2. Brunton, Lauder: Preliminary Note on the Possibility of Treating Mitral Stenosis by Surgical Methods, *Lancet* 1: 352, 1902.
3. Jeger, Ernst: Die Chirurgie der Blutgefäße und des Herzens, Berlin, 1913, A. Hirschwald.
4. Litvak, R.: To be published.
5. Satinsky, V. P.: To be published.
6. Harken, D. E., Ellis, L. B., Ware, P. F., and Norman, L. R.: The Surgical Treatment of Mitral Stenosis, *New England J. Med.* 239: 801-809, 1948.
7. Cossio, P., and Perlman, I.: Cited in Foreign Correspondence, *J. A. M. A.* 137: 659, 1948.
8. Sweet, R.: Unpublished data.
9. Harken, D. E.: Op. cit., p. 802.
10. Bing, R. J.: Personal communication.
11. Taussig, H. B.: Congenital Malformations of the Heart, New York, 1947, Commonwealth Fund.
12. Cutler, E. C., and Levine, S. A.: Cardiectomy and Valvulotomy for Mitral Stenosis, *Boston M. & S. J.* 188: 1023-1034, 1923.
13. Allen, D. S., and Graham, E. A.: Intracardiac Surgery—A New Method, *J. A. M. A.* 79: 1023-1030, 1922.
14. Souttar, P. W.: The Surgical Treatment of Mitral Stenosis, *Brit. M. J.* 2: 603-606, 1925.
15. Smyth, H. G.: An Approach to the Surgical Treatment of Chronic Valvular Disease of the Heart, XVI Annual Assembly of the Southeastern Surgical Congress, April 5-8, Hollywood, Florida, 1948.
16. Cutler, E. C., Levine, S. A., and Beck, C. S.: The Surgical Treatment of Mitral Stenosis; Experimental and Clinical Studies, *Arch. Surg.* 9: 680-821, 1924.
17. Pribram, B. O. A.: Die Operative Behandlung des Mitralstenose, *Arch. f. klin. Chir.* 142: 458-465, 1926.
18. Cutler, E. C., and Levine, S. A.: Op. cit.
19. Harken, D. E.: Op. cit.
20. Satinsky, V. P.: Thoracoabdominal Approach for Portocaval Anastomosis, *Ann. Surg.* 128: 938-947, 1948.
21. Allen, D. S., and Graham, E. A.: Op. cit. 21: 163-171, 1941.
22. Wolff, L., and Levine, H. B.: Hemoptysis in Rheumatic Heart Disease, *Am. Heart J.*
23. Levine, S. A.: Clinical Heart Disease, ed. 2, Philadelphia, 1940, W. B. Saunders Company, p. 68.
24. Bailey, C. P.: The Surgical Treatment of Mitral Stenosis (Mitral Commissurotomy), *Dis. Chest* 15: No. 4, 1949.

DISCUSSION ON "THE SURGICAL TREATMENT OF MITRAL STENOSIS" BY DR. DWIGHT E. HARKEN, DR. LAURENCE B. ELLIS, AND DR. LEONA R. NORMAN;* AND "THE SURGERY OF MITRAL STENOSIS" BY DR. CHARLES P. BAILEY, DR. ROBERT P. GLOVER, AND DR. THOMAS J. E. O'NEILL

DR. RICHARD H. SWEET, Boston.—I have a suggestion to make, referred to by Dr. Bailey, on a procedure which might be of some interest to those of us who have not yet perfected our technique for intracardiac surgery, and I might add that, so far as I am concerned, I am going to wait for Dr. Harken and Dr. Bailey to do that before I start myself.

(Slides.) This operation is an attempt to reproduce outside the heart the physiologic effect of an interauricular septal defect. The diagram on this slide shows what we have done. Before this work was done, Dr. Bland of our Department of Cardiology suggested that in cases of tight mitral stenosis an extracardiac shunt between the pulmonary and the systemic venous systems might provide a sufficient release of pressure to relieve the congested pulmonary circulation.

This slide shows the anatomic findings at the operation on one of the patients. Here is the superior segment of the right lower lobe and at this point can be seen the superior

*See page 1 for article by Harken, Ellis, and Norman.

segmental branch of the inferior pulmonary vein. In this slide, drawn from the same operation after further dissection, there is as you see a small anomalous vein which lies somewhat above the superior segmental vein. In all the patients whom we have operated upon to date the diameter of the superior segmental vein has been equal to that of the azygos vein. My reason for using the nonsuture anastomosis technique is that the vessels are fragile, and it is my opinion that a suture anastomosis might be difficult and not as satisfactory from the functional point of view. At the completion of the operation in this case there was a rush of blood through the anastomosis into the azygos vein and a definite thrill could be felt. There was also a definite drop in the interauricular pressure from 530 mm. of water before the anastomosis was made to 379 mm. of water after the anastomosis was made.

Three of the patients have been enormously improved as a result of the operation. Each of these was formerly committed to a chair and bed existence but now leads an essentially normal existence. The fourth patient's operation was too recent for evaluation of the result. In the fifth case the patient died.

Dr. Harken was searching in his own mind for some objective evidence of striking relief—perhaps we should not call it cure—and I have discovered perhaps the test which he needs. Two of my patients before operation were unable to indulge in sexual intercourse without suffering pulmonary edema, and afterward everything was all right.

DR. A. J. GRACE, London, Ontario.—I have listened spellbound to this presentation by both our colleagues and by Dr. Sweet, who has done excellent work in this as well as other fields. The subject has interested Dr. L. D. Wilcox and myself in London for some years. We also have done a number of experiments on dogs such as have been mentioned. Perhaps two or three points might be mentioned very briefly. In the first place, this work confirms the principles Dr. Harken has given; they are very sound and, I think, in the main basic. One point we might add is that any damage done to one or more chordae tendineae in these operations is likely to lead very rapidly to cardiac dilatation and death within twenty-four hours in most cases. I think that point should be stressed. In all surgical work we seek to avoid adding to the damage and the risks as well as actually contributing to the health of the individual.

One point with regard to pressure changes: Immediately following section of a cusp of the mitral valve the pressure in the auricle rises very rapidly, as Dr. Harken has emphasized, and that provides mechanical problems and an extra danger of bleeding. It is rather amazing how slowly the rise appears in the pulmonary arterial side when records are kept, and how slight it is. Turning to the problem in man: we are dealing with distorted valves, calcified and changed as well as constricted, as the diagrams shown this morning have brought out. We have felt, for the reasons Dr. Harken has given, that the posterior cusp would be a good site for surgical attack; the anterior one has other functions to fulfill in directing blood to the aortic outlet. We have chosen the posterior cusp, but have been impressed by the fact that it is seldom more than one-half the total area of the anterior cusp, and is often less than that in the pathologic material studied in several centers, particularly in these mitral stenotic cases we are talking about. We have sought some means of dividing that calcified cusp, the margin not being divisible by any ordinary cutting edge; it is so resistant in many cases. We have sought a means of dividing that edge, preferably right back into the posterior cusp toward the base, to relieve the pulmonary and left auricular hypertension, to increase the ventricular inflow of blood during diastole, and we hoped that constriction of the base of the ventricle during systole would help to limit regurgitation. We have sought to do this without any loss of valve substance. That is the aim and the principle desired.

A method which we have not yet worked out as fully as we should like, but which may deserve further study, and we feel it does, came to mind from some basic work long ago by Sosman and Christian, who demonstrated quite a marked vertical motion of the heart fluoroscopically in cases where the calcified mitral valve could be observed. My colleague, Dr. Wilcox, demonstrated the same in a patient with a calcified annulus fibrosis, a marked vertical movement of the mitral ring and valve. To be sure this is not as great in extent as in the calcified aortic valve, but it is considerable.

The idea struck us that this motion might be used to advantage if one could pass a cutting type of instrument, such as a section of Gigli saw, down from above, through the left auricular appendage, or one of the pulmonary veins, and out through the apex of the heart. (Slide.) Both ends of the Gigli saw blade had been smoothed off in advance, or they may be encased in close-fitting segments of ureteral catheter. The middle section alone remains as a saw edge and this is maintained in close contact with the margin of the valve cusp to be sectioned by means of a combination of lengthwise traction on the ends of the saw, maintaining tension, and pressure exerted from the outside of the heart at the auriculoventricular ring. The principle is to maintain pressure of the valve cusp against the tense sawing edge, letting the cardiac motion do the actual sectioning of the valve. This technique causes very little blood loss and it does not disturb the cardiac action to any extent provided that the organ is not displaced greatly.

So far this method has not had adequate trial in human cases. We have had opportunity to use it only once. In retrospect, that patient was not truly suitable and little was gained. It is felt that this is simply a reflection of the difficulty in selecting really suitable cases. There is great need of a procedure which will enable us to tell preoperatively the size and condition of the cusps of the mitral valve and the state of the whole mitral ring.

Finally, it is suggested that during actual manipulation of rheumatic hearts, momentary compression of both carotid arteries in the neck by the anesthetist or an assistant may prevent cerebral embolic complications by fragments of valve or blood clot.

DR. O. C. BRANTIGAN, Baltimore.—All those who have contributed to this problem in the past certainly deserve great credit and surely the extensive experimental work has made possible the thoughts Dr. Bailey has upon this subject. I believe one should realize that he has made one very definite contribution, that is, the restoration of a functioning valve, this being obtained by dividing in the normal commissure and, particularly, in the commissure that is in the posterolateral direction so that the anterior leaf is in no way divided or injured. He has done away with the idea of a tunnel because the mechanical rigid tunnel makes hemorrhage control very difficult. By insertion of a finger through an opening of proper size in the auricular appendage very little blood is lost; if the opening is made too large a previously placed purse-string suture can be tightened enough to prevent bleeding. The finding of a normal commissure is done by digital palpation and not by guess, and if one will examine gross autopsy specimens or cadavers one will find this normal commissure is in a pretty definite location; it is difficult to find a true edge of the valve leaf but one can find between the chordae tendineae and papillary muscles a definite location for the commissure. In my experience in examining autopsy specimens and cadavers it is in a pretty definite location. With the cadaver in the usual operating position and the right index finger inserted into the auricular appendage the posterolateral commissure will be at about the medial aspect of the terminal phalanx of the finger. In the stenosed valve the finger, of course, will not pass through the opening, but the commissure location will be fairly definite.

I have recently had the pleasure of watching Dr. Bailey do one of his operations, the entire time consumed being very short, and the blood loss actually not more than two ounces. Following operation there was no murmur to be heard.

DR. DWIGHT E. HARKEN, Boston.—I was interested in Dr. Bailey's optimistic impression of our prospective 5 per cent mortality. I think that is a bit optimistic for 1949 but I believe that we can think of the reverse picture; namely, that there is a malignant disease under discussion. This malignant form of mitral stenosis is being more clearly defined. As we become better acquainted with mitral stenosis, we recognize a picture of hemöptysis, right ventricular failure, and gravely elevated intrapulmonic pressure (by catheterization) that gives us the picture of a malignant disease. It is perhaps more than an interesting coincidence that the only two patients who rejected surgery did die within three months after this refusal.

How to find the mitral orifice has been a problem that has bothered Dr. Bailey as much as it has bothered me. I believe the tunnel helps us. It is flexible and not associated with much clotting. I do not agree with Dr. Brantigan that the mitral ring is in a

constant position. It is not constant because there may or may not have been a prior period of regurgitation with ventricular hypertrophy. There may be varying degrees of pulmonic arterial dilatation and therefore the position of the mitral ring and also the position of the whole left auricle may vary greatly. The position and size of the appendage and also the mitral ring vary so much that in some instances it cannot be approached anterior to the phrenic nerve when the pericardium is opened. Even the appendage may lie posteriorly and require pericardial incision back of the phrenic nerve. These variations must be borne in mind.

Dr. Sweet's comments are timely. His results are extremely good. I hope that we can in time differentiate between patients who should have valvuloplasty and those who should have left to right shunts created.

I did not mean to imply today nor in previous publications in 1948 that I have wanted or accepted regurgitation. Even last year's publications repeatedly used the term "valvuloplasty" and "excision of the commissural zone and ring." A reprint of that paper was sent to Dr. Bailey at that time. One needs only to understand the function of the mitral valve by direct palpation in the laboratory animal to advocate valvuloplasty. As the finger is directed through the mitral ring of the animal, one discovers that the small posterior leaflet has little function and the area is closed by the ventricular systole, whereas the anterior leaflet in systole constitutes a wall that deflects or watersheds blood out through the aorta. Dr. Grace has very nicely emphasized this same point. As Dr. Grace also pointed out, and I thought my diagrams confirmed, the fishmouth opening in the funnel is quite eccentric; it is well back of the midpoint. I believe that point was not brought out in Dr. Bailey's report. These funnels are eccentric, the watershed is anterior, and the commissures are posterolateral and posteromedial. The mouth itself is rigid, the commissures are solidly closed, and the leaflets themselves are very flexible in the vast majority of instances. This concept of the pathologic process is fundamental.

In closing, I would again like to point out the fact that we are dealing with a malignant disease, the recognition of which may become clearer in time. Patients are not only to survive operations but be improved thereby. I hope that our surgical mortality becomes less formidable before we have another meeting.

DR. BAILEY.—It is evident from Dr. Sweet's report that life can be comfortably prolonged in many very seriously ill patients by the shunt operation which he has described, and I believe such an operation will always have a definite place in the surgical armory for the relief of mitral stenosis. However, it seems to me that it is not as good a procedure as one directed at the mitral valve itself, and when such direct surgery is perfected, shunt operations will find a very limited field of usefulness.

Dr. Harken and I may differ in terminology. It was my impression that he believed that a limited regurgitation or directed regurgitation was necessary for the relief of mitral stenosis. He performed a commissurotomy in his patients with a punch, which I believe was not large enough to cut all the way through the scar tissue and therefore did not produce a restoration of valve function. His method as described, however, is certainly a great improvement over the older procedures which produced free regurgitation.

I would like to point out that it is very easy to explore the valve and its commissures with the finger inserted through the auricular appendage. Because of the excessive left auricular pressure resulting from the mitral stenosis, the left auricular appendage and auricle are large and lend themselves to very adequate exploration with the finger. By the use of a purse-string suture about the base of the appendage, bleeding can be very readily controlled and is not serious. The purse-string is tied around the finger while it is in place within the appendage, so that no blood escapes during this part of the procedure.

Dr. Grace has pointed out that some of these valves are very calcified. We believe that in such cases if it is not possible to cut the commissures with the knife, it is possible to relieve the obstruction with a punch. For this purpose we have devised a narrow long-bladed backward-cutting forceps or punch which actually accomplishes the effect of commissurotomy even though it removes a small sliver of valvular tissue at the commissures.

At the present time we have formulated certain contraindications and indications for this procedure. We believe that active rheumatic infection, the presence of frank decompensation, and multivalvular lesions are contraindications. If the left ventricle is enlarged, one can be fairly certain that there is some other associated lesion and not pure mitral stenosis alone. Such lesions may include marked mitral regurgitation or aortic stenosis and regurgitation. The presence of subacute bacterial endocarditis and lesions in patients over the age of 40 years are further contraindications. We feel that the ideal indication for commissurotomy is (pure) mitral stenosis with no, or very early, symptoms, provided the pressure in the right ventricle and pulmonary artery can be demonstrated to be elevated. If pulmonary hypertension has begun to develop, these patients will undoubtedly run into serious cardiac difficulties within two to three years. If they are operated upon before serious symptoms have developed, not only can the valve be surgically treated more effectively, but the ultimate result will also be much improved. If a patient has had a previous bout of cardiac decompensation but can be satisfactorily restored to compensation by medical means, such a patient may still be subjected to surgery and a good result obtained. After further experience it is possible that future indications might include cases complicated by other valvular lesions, and possibly even those who are in mild decompensation, no further improvement under medical management being obtainable.

ACIDOSIS DURING THORACIC SURGERY

HENRY K. BEECHER, M.D., AND ANNA J. MURPHY, B.S. (BY INVITATION)
BOSTON, MASS.

THE chain of events that leads to success or failure in thoracic surgery is more complex than it is in other fields. Notwithstanding the progress made in recent years, the occasional patient succumbs near the end of operation or shortly thereafter, or more often barely pulls out of a puzzling condition of low blood pressure. At such times it is customary to say, "The blood replacement was not sufficient," or, "His heart just would not stand it," and so on. Stand what is often not clear.

The purpose of this paper is to present factual data that explain some of the unsatisfactory experiences mentioned. We shall describe an acidosis of potentially fatal degree that results from accepted procedures in thoracic surgery, an acidosis that increases smoothly and swiftly with the passage of time.

POSITION OF PATIENTS AND ANESTHESIA PROCEDURES

Our anesthesia procedures have been described in detail elsewhere.^{2, 3} Briefly, our patients are operated upon with the diseased side vertically uppermost with a blanket roll or pillow of approximately five inches in diameter placed beneath the thorax. About 5 degrees of Trendelenburg position is used, and then the operating table is broken 10 to 15 degrees at the head and 5 degrees at the foot.

Preanesthetic medication consists in most cases of morphine, 10 mg., and atropine, 0.6 mg., subcutaneously. Unconsciousness is induced with 80 volumes per cent nitrous oxide with 20 volumes per cent oxygen, and then the shift is made to straight ether maintenance with 95 volumes per cent oxygen in a closed system, usually with "circle" soda lime filter. Endotracheal tubes are always used, Flagg, size 8, or Magill, 32 French. Inflatable cuffs are not used on the endotracheal tubes employed in these cases. (We do sometimes use them in transthoracic esophagectomy where the positive pressure needed can cause troublesome gastric distention.) Positive pressure is used when necessary to provide normal oxygenation of the blood, to reduce mediastinal swing, and to inflate the lungs at about twenty-minute intervals. Aspiration of the bronchi is carried out to maintain a clear airway.

CHEMICAL METHODS

Samples.—Blood was drawn from the radial artery in oiled syringes and transferred under oil. Heparin was used to prevent coagulation in that part of the blood used for the oxygen determination. Carbon dioxide and pH were measured in serum. The first blood sample was drawn immediately before the induction of anesthesia while the patient breathed room air, and subsequent samples were drawn while the patient was connected with the closed anesthesia apparatus.

From the Anesthesia Laboratory of the Harvard Medical School at the Massachusetts General Hospital.

Read at the Twenty-ninth Annual Meeting of The American Association for Thoracic Surgery, New Orleans, La., March 29-31, 1949.

Oxygen.—Oxygen was determined by the method of Van Slyke and Neill.¹³ When the blood contained ether, the Snyder¹⁴ modification of the Shaw and Downing¹² method was used. In this procedure the oxygen and carbon dioxide, after extraction from the blood, are stored in a pipette attached to the machine while the vacuum chamber is cleaned. Water is substituted for the ferrieyanide, the gases returned to the chamber, and the analysis continued as in the original method. One cubic centimeter samples were used. Oxygen capacities were determined on all samples by aerating the blood in room air for about five minutes.

Carbon Dioxide.—Carbon dioxide determinations were made on 0.5 c.c. serum by the Van Slyke and Neill method.¹³ When ether was present in the blood, the Austin¹ method was used with some modification, as follows: The serum is acidified and extracted in the usual manner except that one very small drop of caprylic alcohol is used instead of the usual two drops. The p_1 is read, the extraction is repeated, and p_1 is read again. The mercury is allowed to rise in the chamber to the 2 c.c. mark at the same rate as in an analysis without ether. The carbon dioxide is absorbed with 0.25 c.c. of 5 N NaOH, and p_2 is read. The mercury is lowered to the 50 c.c. mark, and the chamber is shaken for three minutes. The volume is reduced to 2 c.c., and p_3 is read. This is repeated until p_3 is constant. If there is sufficient caprylic alcohol to prevent foaming but not enough to form a layer above the solution in the vacuum chamber, then p_1 and p_3 readings will check themselves within 0.5 mm. The volume of the acidified solution extracted was 3.5 c.c. when 0.5 c.c. of serum was used. The K for the machines under these conditions was 0.64. The formula used $[CO_2] = [p_1 - p_3 + K(p_3 - p_2)] \times f$.

Carbon Dioxide Tension.—The carbon dioxide tension was calculated from the carbon dioxide content (volumes per cent) and the pH value. In most cases this was read off the Peters and Van Slyke chart.¹⁰ When the values were beyond the limits of this, the Henderson-Hasselbalch equation was used directly to calculate the tension,

$$P = \frac{CO_2}{0.1316 \times 0.510 (10^{pH - pK^1 + 1})}$$

When the pH is in the neighborhood of 7.0 it is not possible to estimate the carbon dioxide tension with as great accuracy as when the pH is higher, since small changes in pH in the low region cause relatively large changes in the carbon dioxide tension.

Hydrogen-ion Concentration.—Hastings and Sendroy⁹ phenol red bicolor standards for studies in acidosis were used for the pH: 0.2 c.c. of serum were added to 4.0 c.c. of saline phenol red solution previously adjusted to 7.45. Then 0.2 c.c. serum in 4.0 c.c. saline solution was used as a blank. These were warmed to 38° C., placed in the colorimeter block, and matched to the nearest set of standards. The standards can be read for every 0.05 change in the pH and the final reading on the serum can be estimated to 0.02 pH.

Hematocrit Value.—Hematocrit value was determined on heparinized blood in Rourke-Ernstene¹² tubes which were spun at 2,500 r.p.m. for forty-five minutes.

RESULTS

The principal data obtained in 43 patients, in two age groups, are given in Tables I to VI. They are summarized in Table VII, A in order to compare the patient's state near the end of operation with what it was at the beginning. The average data in Table VII, A show that while the carbon dioxide content remained constant, the pH decreased sharply in the lobectomy and pneumonectomy groups. These findings in corrected form, in conjunction with the calculated increases in fixed acid shown in Table VII, B, indicated that these patients were in a rather severe acidosis of combined respiratory and metabolic types.

Table VIII shows that the rise in alveolar carbon dioxide tension began and developed to a significant degree before the pleura was opened. And finally, Table IX demonstrates that the anesthetic agent or apparatus was not responsible for the rise in carbon dioxide tension. Table X illustrates how assisted respiration was effective in lowering the carbon dioxide tension in one case but not in another.

Attention is called to the fact that the oxygen tensions shown in these tables are generally satisfactory, even near the termination of pneumonectomy, and at a time when the carbon dioxide tensions are dangerously high.

There is some suggestion in the pneumonectomy group that the hematocrit rises during operation, but an examination of the standard errors of the means does not indicate significance in the available data.

DISCUSSION

In the normal, unanesthetized man an increase in the concentration of carbon dioxide in the inspired air results in an increase in pulmonary ventilation, until about 9 per cent is reached. Further increase in carbon dioxide causes depression of the respiration,¹¹ presumably owing to the well-known anesthetic properties of the gas.

Under our present circumstances no such respiratory response to the high tensions of carbon dioxide was evident, probably because of the depressing action of ether on the respiratory center.⁶

It is quite clear from Fig. 1 that under present circumstances (unlike the normal), respiration which is adequate to provide oxygen is not adequate to remove carbon dioxide. Preoccupation with color of a patient's blood, important as this is, is not enough. This is only half of the problem. The carbon dioxide level must also be considered. The tensions of this gas reached in some of our patients might of themselves be fatal, particularly in patients who had been subjected to considerable blood loss and surgical trauma.

Why the carbon dioxide tension should rise so high must be considered. This is not the usual effect of ether with patients in the supine position (see Table IX). Presumably this great rise is to be blamed on position and the open

pleura and the depression of respiration by the anesthetic. Some facts to be examined in this connection are the following.

In eleven patients (Table VIII) the alveolar carbon dioxide tension was determined immediately prior to the induction of anesthesia and then some forty-two minutes later, but before the pleura was opened. (These patients had been in the operative position with the diseased side uppermost for approximately twenty minutes of this time.) Ten of these eleven patients showed a rise of 14.6 mm. Hg or 2.0 volume per cent, average. Thus, the alveolar carbon dioxide tension had begun to swing up sharply before the pleura was opened and while the tidal volume and minute volume of respiration appeared to be quite normal. The open pleura is therefore not the only factor responsible for the later rise in carbon dioxide tension.

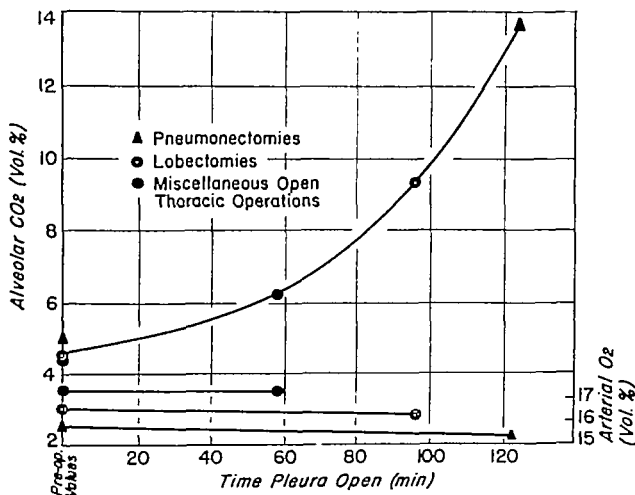


Fig. 1.

It must be emphasized that this increase in alveolar tension of 14.6 mm. Hg., or 2.0 volume per cent, is a truly great rise. It has been found (Campbell, Douglas, Haldane, and Hobson⁶) in the average individual that a rise in pressure of 1.6 mm. Hg will double pulmonary ventilation. In the normal unanesthetized man a rise of only 1.0 volume per cent in the alveolar carbon dioxide content is sufficient to increase the pulmonary ventilation about 50 liters per minute.

How can this early rise in alveolar tension of carbon dioxide be explained? A number of factors are present: besides the position, we must consider the increase in dead space produced by the anesthesia apparatus, the effect of the endotracheal tube, and possibly the effect of the anesthetic agent on the bronchiolar musculature. Conceivably an increase in dead space could be caused by the

TABLE I. PNEUMONECTOMIES

PATIENT	AGE (YR.)	DURA- TION OF ANES- THESIA (MIN.)	PLEURA	TIME AFTER ANES- THESIA STARTED (MIN.)	TIME AFTER PLEURA OPENED (MIN.)	O ₂ CONTENT (VOL. %)	O ₂ CAPACITY (VOL. %)	% SATURA- TION	CO ₂ CONTENT (VOL. %)	pH	ALVEOLAR CO ₂ TENSION	HEMA- TOCRIT % CELLS	REMARKS
B. W.	49	190	Adherent	0	0	12.6	12.8	99	56.8	7.44	37.4	34.2	March 25
				30	0	14.0	12.5	89	55.5	7.40	39.5	36.9	March 25
				75	25	15.3	13.9	91	62.5	7.27	59.5	39.0	March 25
				168	118	14.0	14.3	98	62.3	7.07	90.0	42.8	March 25
C. B.	57	260	Adherent	0	0	17.2	17.4	99	52.0	7.55	26.3	45	Feb. 16
				230	180	15.7	17.4	90	72.3	7.03	115.8	46	Feb. 16
				246	196	17.3	17.4	99	68.5	7.08	96.7	46	Feb. 16
						18.7	18.9	99	56.5	7.45	36.0	44	Feb. 17
W. J.	54	200	Adherent	0	0	12.4	13.5	91.7	56.5	7.50	33.3	34.5	Feb. 12
				30	0	14.2	13.4	100	65.3	7.15	84.0	37.3	Feb. 13
				90	50	13.2	13.5	97.7	61.8	7.15	82.5	37.8	Feb. 13
				175	135	15.5	15.5	100	71.4	7.08	100.6	43.2	Feb. 13
H. V.	41	170	Adherent	0	0	14.1	16.3	86.5	64.6	7.40	48.0	42.6	Feb. 17
				40	0	17.1	17.5	97.7	55.7	7.45	36.5	39.7	March 13
				128	83	18.8	19.1	98.5	57.6	7.28	53.0	44.4	March 13
				175	130	17.8	18.0	98.8	52.5	7.25	60.0	42.8	March 13
L. T.	18	120	Adherent	0	0	14.1	14.7	95.7	60.2	7.57	30.3	35.2	March 18
				38	0	15.2	16.4	92.7	63.9	7.55	32.5	41.1	Dec. 18
				85	40	15.9	16.3	97.5	61.5	7.35	49.0	46.6	Dec. 19
				130	85	14.1	15.5	91.0	67.5	7.15	84.0	46.6	Dec. 19
C. B.	36	190	Adherent	0	0	10.3	14.1	73.2*	70.9	7.00	133.1	44.7	Dec. 19
						17.2	17.1	100	66.0	7.50	37.5	43.8	Dec. 24
						16.2	16.8	97	63.2	7.57	30.7	44.0	Dec. 27
						17.4	17.9	97.2	59.7	7.50	33.9	43.8	Feb. 17
E. L.	24	230	Free	0	0	18.0	18.5	97.3	62.4	7.45	39.5	43.7	Sept. 20
				40	0	18.0	18.5	97.2	61.4	7.45	39.0	43.1	Sept. 22
				93	15	18.7	19.8	95	69.8	7.18	79.5	52.2	Sept. 22
				200	144	16.3	17.0	96	59.5	7.45	38.0	44.3	Sept. 22
E. L.	24	230	Free	0	0	13.2	14.5	91	57.4	7.45	33.8	36.9	Sept. 23
						17.1	18.5	92.5	59.0	7.55	30.0	45.8	Sept. 30
				40	0	20.8	20.6	100	60.6	7.46	37.3	52.2	May 15
				93	37	18.1	19.8	91.5	55.3			58.4	May 16
E. L.	24	230	Free	200	144	17.2	18.4	93.5	58.2			54.0	May 16
						18.5	18.6	100	56.7			50.0	May 17

B. H.	13	170	Adherent	0 40 120 155	0 0 77 112	12.6 18.1 17.8 16.9	14.5 17.7 17.3 16.7	86.9 100 100 100		7.48	34.5	43.4 50.0 45.0 48.6 53.3	May 22 May 23 May 23 May 23 May 26
M. F.	34	170	Adherent	0 72 117 157	0 0 35 75	13.3 14.2 14.4 14.6	14.7 15.3 14.5 16.2	90.5 93 100 96	59.8 59.7 56.8 53.2	7.45	37.8	37.5 41.2 38.7 40.4	July 2 July 2 July 2 July 2
L. M.	19	220	Adherent	45 105 220	0 40 155	19.5 18.6 17.7			52.7 62.6 50.5			48.3 47.9 45.2	July 26 July 26 July 26
P. F.	49	240	Adherent	36 92 144 182 217	0 46 98 136 171	17.0 16.4 15.2 15.6 16.4			63.0 60.5 68.9 67.2 61.3			47.1 47.9 45.9 40.1 47.8	July 29 July 29 July 29 July 29 July 29
R. D'E.	34	190	Adherent	27 40 87 108 190	0 0 14 57 139	16.8 13.5 12.4 13.8			42.3 46.1 49.6 50.3			45.0 45.8 47.0 48.4 54.7	April 3 April 3 April 3 April 3 April 3
T. F.	28	170	Adherent	65 110 160 178	0 37 87 105	16.0 16.1 12.9 13.0			58.2 58.0 54.7 50.3			41.0 41.4 40.9	April 17 April 17 April 17 April 17
J. D.	52	230	Adherent	35 95 146 103 140 230	0 42 93 140 177	21.1 20.1 19.5 17.7			68.2 64.3 65.7 60.6 57.1			49.5 47.9 49.0 46.4 47.6	July 10 July 10 July 10 July 10 July 10
W. N.	23	200	Adherent	67 97 142	0 25 70	18.7 16.3			58.4 58.3 60.3				Dec. 22 Dec. 22 Dec. 22
C. H.	00	180	Free	49 63 115 150-161	0 4 56 91-102	10.1 13.0 12.1			73.8 75.3 72.8 74.8			Exceed- ingly high, above 70%	Feb. 15 Feb. 15 Feb. 15 Feb. 15

*Floods of pus in airway.

K.K.	54	160	Free	0	0	16.4	16.5	100	61.4	7.55	31.5	41.7	Oct. 8
				38	0	19.3	17.9	100	60.8	7.37	40.5	42.9	Oct. 8
				86	24	19.1	17.7	100	60.6	7.22	72.5	45.0	Oct. 8
				132	70	19.1	17.6	100	65.9	7.18	76.0	47.1	Oct. 8
F.S.	55	255	Adherent	0	0	17.9	18.3	97.9	59.7	7.55	31.5	48.0	Oct. 28
				35	0	20.7	19.0	100	60.0	7.30	60.5	52.3	Oct. 28
				130	08	19.1	18.7	100	68.6	7.10	88.0	52.5	Oct. 28
				228	166	18.1	18.8	96.3	68.1	7.05	103.0	51.1	Oct. 28
R. M.	31	190	Adherent	0	0	18.0	18.9	95.3	48.8	7.50	37.8	44.4	Dec. 31
				40		17.8	17.5	98.5	58.0	7.45	30.0	45.0	Dec. 31
				105		16.6	16.8	98.7	53.5	7.20	62.0	44.3	Dec. 31
				190		16.3			49.0	7.10	68.0	45.3	Dec. 31
R. R.	55	150	Free	0	0	14.7	15.6	94.2	55.7	7.47	34.7	37.2	Feb. 9
				44	0	17.7	17.8	100	59.2	7.28	54.0	44.8	Feb. 9
				91	44	17.2	16.8	100	59.8	7.28	58.0	44.5	Feb. 9
				132	85	17.8	16.7	100	62.3	7.28	58.0	45.0	Feb. 9
K.P.	34	240	Adherent	0	0	16.3	17.6	93	65.2	7.53	34.4	43.4	Nov. 12
				55	0	18.4	17.5	100	60.0	7.45	37.8	45.2	Nov. 13
				110	45	19.1	18.4	100	63.6	7.38	46.5	40.7	Nov. 13
				180	115	18.7	17.7	100	60.7	7.37	46.0	48.9	Nov. 13
						15.9	15.8	100	62.3	7.47	38.0	43.6	Nov. 14
						18.7	19.6	95	62.3	7.55	32.0	48.4	Jan. 6
Y. B.	30	120	Adherent			16.8			56.3				Oct. 9
						13.9			63.5				Oct. 9
J. P.	28	210	Adherent	45	0	18.0			51.3			43.0	July 19
				96	46	18.9			53.7			43.5	July 19
				184	134	18.9			47.9			44.1	July 19
				225	175				40.7			48.0	July 19
E. K.	37	145	Adherent	40	0	17.2			50.4				Oct. 6
				93	48	14.5			67.9				Oct. 6
				119	74	14.4			57.5				Oct. 6
E. B.	47	170	Free	50	0	15.3			57.6				Oct. 13
				90	30	15.2			50.2				Oct. 13
				120	60	14.6			60.0				Oct. 13
				160	90	14.8			56.0				Oct. 13
P. S.	15	160	Free	47	0	13.8			43.0				Oct. 25
				122	67	14.6			49.0				Oct. 25
V. B.	16	240	Adherent	50	0	10.8			52.9				Nov. 16
				155	95				51.1				Nov. 16
F. D.P.	27	150	Adherent	62	0				53.8				Feb. 16
				60	0	18.3			51.9				Feb. 16
				105	30	18.1			50.2				Feb. 16

TABLE III. MISCELLANEOUS

PATIENT	AGE (YR.)	OPERATION	DURATION OF ANESTHESIA (MIN.)	PLEURA	TIME AFTER ANESTHESIA STARTED (MIN.)	TIME AFTER PLEURA OPENED (MIN.)	O ₂ CONTENT (VOL. %)	O ₂ CAPACITY (VOL. %)	% SATURATION	CO ₂ CONTENT (VOL. %)	pH	ALVEOLAR CO ₂ TENSION	HEMATOCRIT % CELLS	REMARKS
E. A.	64	Inoperable cancer	115	Free	0 90	0 40	15.2 15.7	15.9 16.0	96 98	57.4 56.4	7.48 7.43	34.0 37.0	38 39	Feb. 11 Feb. 11
E. D.	52	Excision of cyst of mediastinum	130	Adherent	0 55 135	0 0 70	17.0 18.4 17.3	17.8 18.3 16.9	95.5 100 97.6	59.2 57.9 56.4	7.55 7.45 7.45	30.5 36.6 36.3	43.3 43.7 40.4	Dec. 29 Dec. 29 Dec. 29
G. G.	31	Enucleation of tumor	160	Free	0 50 115	0 0 55	18.4 17.4	18.6 17.5	98.9 99	60.1 51.3 55.2	7.53 7.55 7.42	31.9 26.3 37.8	47.4 48.3 47.8	Oct. 15 Oct. 17 Oct. 17
E. L.	60	Inoperable exploratory thoracotomy	55		0 38 57	0 0 12	15.0 17.0 16.9	16.7 17.7 16.8	89.7 100 100	57.8 61.9 61.3	7.55 7.30 7.26	28.8 54.0 58.1	44.1 48.0 48.0	Oct. 15 Oct. 16 Oct. 16
I. L.	42	Excision of cyst of mediastinum	180	Free	41 88 145	0 38 95	23.1 20.6 21.8			57.3 59.1 58.7			53.6 52.0 51.5	Apr. 22 Apr. 22 Apr. 22
R. T.	55	Inoperable carcinoma of right lower lobe	80	Free	27 42 55	0 10 23	17.1 16.0 15.0			55.5 60.4 56.5			43.1 44.5 43.7	March 25 March 25 March 25
W. B.	27	Segment left lower lobectomy and lingulectomy	160	Adherent	0 35 88 150	0 0 45 107	17.8 18.6 17.0 18.6 20.6 19.0	18.9 20.9 18.8 20.0 19.4 19.1	94.3 100 90.4 93.0 100 100	59.2 57.4 56.9 57.3 56.0 61.1	7.47 7.32 7.34 7.30 7.43 7.43	36.0 49.0 47.0 50.5 37.0 40.0	45.8 47.0 50.6 51.2 44.2 50.0	Sept. 24 Sept. 24 Sept. 24 Sept. 24 Sept. 24 Sept. 25

TABLE IV, A. PNEUMONECTOMIES

PATIENT	SEX	AGE (YR.)	BLOOD BEFORE OPERATION			BLOOD AT END OF OPERATION			BLOOD DAY FOLLOWING OPERATION		
			CO ₂ CONTENT (VOL. %)	pH	ALVEOLAR TENSION CO ₂ (mm.Hg)	CO ₂ CONTENT (VOL. %)	pH	ALVEOLAR TENSION CO ₂ (mm.Hg)	CO ₂ CONTENT (VOL. %)	pH	ALVEOLAR TENSION CO ₂ (mm.Hg)
44 Years and Under											
W. N.	♂	23	58.4			60.3					
R. J. E.	♂	34	42.3			50.3					
T. F.	♀	28	58.2			50.3					
L. A. L.	♀	19	52.7			50.5					
E. L.	♂	24	60.6	7.40	37.3	58.2			56.7	7.37	43.0
B. H.	♂	13	58.2	7.48	34.5	59.7			57.8		
M. F.	♀	34	50.8	7.45	37.8	53.2					
C. B.	♂	30	62.4	7.45	39.5		7.00	133.1	57.4	7.48	33.8
L. T.	♂	18	63.0	7.55	32.5	70.0	7.28	50.2	66.0	7.50	37.5
IL. V.	♀	41	55.7	7.45	36.5	52.5			60.2	7.57	30.3
Average			57.2	7.47	36.4	56.2			59.6	7.48	30.2
45 Years and Over											
C. H.	♂	60	73.8			74.8					
J. D.	♂	52	68.2			57.1					
P. F.	♂	49	63.0			61.3					
W. J.	♂	54	50.4	7.50	33.3	71.4	7.08	100.6	61.0	7.40	48.0
B. W.	♂	49	56.8	7.44	37.4	62.3	7.07	90.0			
C. B.	♂	57	52.0	7.55	26.3	72.3	7.03	115.8	56.5	7.45	36.0
Average			61.7	7.50	32.3	66.5					
Grand average			58.0	7.48	35.0	60.3	7.09	97.0	59.0	7.46	38.1

TABLE IV, B. PNEUMONECTOMIES

PATIENT	SEX	AGE (YR.)	DURATION OF ANES- THESIA (MIN.)	BLOOD TAKEN BEFORE OPERATION				BLOOD TAKEN AT END OF OPERATION				BLOOD TAKEN DAY FOLLOWING OPERATION					
				O ₂ CONTENT (VOL. %)	O ₂ CAPACITY (VOL. %)	% SATU- RATION	HEMA- TOCRIT % CELLS	TIME AFTER PLEURA OPENED (MIN.)	O ₂ CONTENT (VOL. %)	O ₂ CAPACITY (VOL. %)	% SATU- RATION	HEMA- TOCRIT % CELLS	O ₂ CONTENT (VOL. %)	O ₂ CAPACITY (VOL. %)	% SATU- RATION	HEMA- TOCRIT % CELLS	
44 Years and Under																	
W. N.	♂	23	260					70	16.3								
R. d'E.	♂	34	190	16.8			45	139	13.8								
T. F.	♀	28	170	16.6			41	105	13.0								
L. M.	♀	19	220	19.5			48	155	17.7								
E. L.	♂	24	230	17.1	18.5	93	46	144	17.2	18.4	93	45	18.5	18.6	99	50	
B. H.	♂	13	170	12.6	14.5	87	43	112	16.9	16.7	100+	49	17.9	17.5	100+	53	
M. F.	♀	34	170	13.3	14.7	91	38	75	14.6	15.2	96	40					
C. B.	♂	36	190	18.0	18.5	98	44	40	10.3	14.1	73	45	13.2	14.5	91	37	
L. T.	♂	18	120	15.2	16.4	93	41	130	17.8	18.0	99	45	17.2	17.1	100	44	
H. V.	♀	41	170	17.1	17.5	98	40						14.1	14.7	96	35	
Average				16.2	16.7	93	43	107	15.3	16.5	92	47	16.2	16.5	97	44	
45 Years and Over																	
C. H.	♂	60	180	10.1				97	12.1								
J. D.	♂	52	230	21.1			50	140	17.7								
P. F.	♂	49	240	17.0			47	171	16.4								
W. J.	♂	54	200	12.4	13.5	92	35	135	15.5	15.5	100	43	14.1	16.3	87	43	
B. W.	♂	49	190	12.6	12.8	99	34	118	14.0	14.3	98	43					
C. B.	♂	57	260	17.2	17.4	99	45	180	15.7	17.4	90	46	18.7	18.9	99	44	
Average				15.1	14.6	97	42	140	15.2	15.7	96	45					
Grand average				15.7	16.0	94	43	122	15.3	16.2	94	46	16.2	16.8	96	44	

TABLE V, A. LOBECTOMIES

PATIENT	SEX	AGE (YR.)	BLOOD BEFORE OPERATION			BLOOD AT END OF OPERATION			BLOOD DAY FOLLOWING OPERATION		
			CO ₂ CONTENT (VOL. %)	pH	ALVEOLAR TENSION CO ₂ (MM.Hg)	CO ₂ CONTENT (VOL. %)	pH	ALVEOLAR TENSION CO ₂ (MM.Hg)	CO ₂ CONTENT (VOL. %)	pH	ALVEOLAR TENSION CO ₂ (MM.Hg)
44 Years and Under											
E. K.	♂	37	50.4			57.5					
Y. B.	♀	30	50.3			63.5					
P. S.	♂	15	43.0			49.0					
V. B.	♀	10	52.9			51.1					
F. dup.	♂	27	51.9			56.2					
D. E.	♀	23	56.2			49.5					
A. I.	♂	18	61.5			50.7					
J. P.	♀	28	51.3			47.9					
E. M.	♂	27	60.6			47.4					
E. C.	♀	12	71.4			61.6					
K. P.	♂	34	65.2	7.53	34.4	60.7	7.37	46.0	61.6	7.47	38.0
H. O.	♂	28	59.8	7.50	34.0	68.3	7.23	69.0	62.3	7.45	41.0
R. M.	♀	31	48.8	7.50	27.8	49.9	7.10	68.0	64.0		
A. A.	♂	40	60.7	7.50	35.8	55.7	7.30	51.5	52.5	7.48	32.0
Average			56.9	7.51	33.0	55.6	7.25	58.6	60.3	7.47	37.0
45 Years and Over											
E. B.	♀	47	57.6			50.0					
G. B.	♂	46	60.1			64.5					
M. N.	♂	59	57.4	7.48	33.8	63.6	7.25	61.0			
R. R.	♂	55	55.7	7.47	34.7	62.3	7.28	58.0			
K. K.	♂	54	61.4	7.55	31.5	65.9	7.18	76.0			
F. S.	♂	55	59.7	7.55	31.5	68.1	7.05	103.0			
Average			58.6	7.51	32.9	63.4	7.19	74.5			
Grand average			57.4	7.51	32.9	57.9	7.22	68.6	60.3	7.47	37.0

TABLE V, B. LOBECTOMIES

PA- TIENT	SEX	AGE (YR.)	DURATION OF ANES- THESIA (MIN.)	BLOOD TAKEN BEFORE OPERATION				BLOOD TAKEN AT END OF OPERATION				BLOOD TAKEN DAY FOLLOWING OPERATION					
				O ₂ CONTENT (VOL. %)	O ₂ CAPACITY (VOL. %)	% SATU- RATION	HEMA- TOCRIT % CELLS	TIME AFTER PLEURA OPENED (MIN.)	O ₂ CONTENT (VOL. %)	O ₂ CAPACITY (VOL. %)	% SATU- RATION	HEMA- TOCRIT % CELLS	O ₂ CONTENT (VOL. %)	O ₂ CAPACITY (VOL. %)	% SATU- RATION	HEMA- TOCRIT % CELLS	
44 Years and Under																	
E. K.	♂	37	145	17.2				74	14.4								
Y. B.	♀	30	120	16.8					13.9								
P. S.	♂	15	160	13.8				67	14.6								
V. B.	♀	16	240	16.8				95									
F. di'P.	♂	27	150	18.3				30	18.1								
D. E.	♀	23	130	18.9			42	83	17.3			40					
A. I.	♂	18	230	23.8			55	143	19.2			53					
J. P.	♀	28	210	18.0		91	44	134	18.6			44					
E. M.	♂	27	190	14.6	16.0		39	119	14.9				12.2	13.0	94	34	
E. C.	♀	12	170	18.1	17.6	100+	47	80	15.5	14.2	100+	49					
K. P.	♂	34	240	16.3	17.6	93	43	115	18.7	15.1	100+	49	15.9	15.8	100	44	
H. C.	♂	23	170	14.9	15.3	97	38	120	15.6	17.7	100+	42	13.5	14.4	94	37	
R. M.	♀	31	190	18.0	18.9	95	44	130	16.3	15.7	99	45					
A. A.	♂	40	150	15.4	16.3	95	40	120	15.8	16.3	97	36	13.8	14.0	100	39	
Average				17.2	17.0	95	44	101	16.4	15.8	99	44	13.9	14.3	97	38	
45 Years and Over																	
E. B.	♀	47	170	15.3				90	14.8								
G. B.	♂	46	190	13.4	14.3		40	48	13.6	14.4	95	42					
M. N.	♂	59	70	14.6	15.5	94	40	48	12.5	15.3	82	41					
R. R.	♂	55	150	14.7	15.6	94	37	85	17.8	16.7	100+	46					
K. K.	♂	54	160	16.4	16.5	99	42	70	19.1	17.6	100+	47					
T. S.	♂	55	255	17.9	18.3	98	48	166	18.1	18.8	96	51					
Average				15.4	16.0	96	41	85	16.0	16.6	95	45					
Grand average				16.7	16.5	95	43	96	16.2	16.2	97	45	13.9	14.3	97	38	

TABLE VI, A. MISCELLANEOUS

PATIENT	SEX	AGE (YR.)	BLOOD BEFORE OPERATION			BLOOD AT END OF OPERATION			BLOOD DAY FOLLOWING OPERATION		
			CO ₂ CONTENT (VOL. %)	pH	ALVEOLAR TENSION CO ₂ (MM.Hg)	CO ₂ CONTENT (VOL. %)	pH	ALVEOLAR TENSION CO ₂ (MM.Hg)	CO ₂ CONTENT (VOL. %)	pH	ALVEOLAR TENSION CO ₂ (MM.Hg)
44 Years and Under											
H. L.	♂	42	57.3			58.7					
W. B.	♂	27	59.2	7.47	30.0	57.3	7.30	50.5	61.1	7.43	40.0
G. C.	♂	31	60.1	7.53	31.0	55.2	7.42	37.8			
45 Years and Over											
R. T.	♂	55	55.5			56.5					
E. L.	♀	60	57.8	7.55	28.8	61.3	7.26	58.1			
E. D.	♀	52	59.2	7.55	30.5	50.4	7.45	36.3			
E. A.	♂	64	57.4	7.48	34.0	50.4	7.43	37.0			
Average			58.1	7.52	32.2	57.4	7.37	43.9			

TABLE VI, B. MISCELLANEOUS

PA- TIENT	SEX	AGE (YR.)	DURA- TION OF ANES- THESIA (MIN.)	BLOOD TAKEN BEFORE OPERATION				BLOOD TAKEN AT END OF OPERATION				BLOOD TAKEN DAY FOLLOWING OPERATION				OPERATION	
				O ₂ CON- TENT (VOL. %)	O ₂ CAPAC- ITY (VOL. %)	% SATU- RATION	HEMA- TOCRIT %	TIME AFTER PLEURA OPENED (MIN.)	O ₂ CON- TENT (VOL. %)	O ₂ CAPAC- ITY (VOL. %)	% SATU- RATION	HEMA- TOCRIT %	O ₂ CON- TENT (VOL. %)	O ₂ CAPAC- ITY (VOL. %)	% SATU- RATION		HEMA- TOCRIT %
44 Years and Under																	
H. L.	♂	42	180	23.1			53.6	95	21.8				51.5				Excision of cyst of mediastinum
W. B.	♂	27	160	17.8	18.9	94	45.8	107	18.6	20.0	93	51.2	19.0	19.1	100	50.0	Segment lower lobec- tomy and lingulectomy
G. C.	♂	31	160	18.4	18.6	99	47.4	55				47.8					Excision of mediastinal tumor
45 Years and Over																	
R. T.	♂	55	80	17.1				23	15.0								Inoperable carcinoma of right lower lobe
E. L.	♀	60	90	15.0	16.7	90	44.1	12	16.9	16.8	100	48.0					Inoperable exploratory thoracotomy
E. D.	♀	52	130	17.0	17.8	96	43.3	70	17.3	16.9	98	40.4					Excision of cyst of mediastinum
E. A.	♂	64	115	15.2	15.9	96	38.0	40	15.7	16.0	98	39.0					Inoperable carcinoma
Average				17.7	17.6	95	45.4	57.4	17.6	17.4	97.2	46.3					

effect of the anesthetic (ether) on these muscles. A check on the role of these factors will be presented forthwith.

Ether, the anesthesia apparatus, and the endotracheal tube can be ruled out. They are of themselves unimportant factors in the rise in carbon dioxide tension as shown in Table IX and elsewhere (Beecher, Francis, and Anfinson⁴). Clearly (Table VIII), position is a factor in the rise of carbon dioxide tension. Other chief factors are the open pleura and depression of respiration by the anesthetic agent. These are the obvious factors. There is a curious increase in fixed acid (as shown in Table VII, B) with the passage of time. Possibly the high tensions of carbon dioxide encountered here interfere with cellular oxidations to the extent that fixed acids accumulate. The accumulation of fixed acid is puzzling, particularly in view of the failure of ether anesthesia alone to do this, as shown elsewhere.⁴ The fixed acid problem is being studied further.

The statement is sometimes made that the answer to all these problems is mechanical respiration. There is perhaps still some room for doubt concerning this. For one thing, the mechanical respirators used with anesthetic agents have

TABLE VII, A. ARTERIAL BLOOD

OPERATION	BEFORE OPERATION				AT OPERATION'S END			
	NO. OF PATIENTS*	O ₂ CONT. (VOL. %)	% SAT.	HEMATOCRIT % CELLS	TIME AFTER PLEURA OPEN (MIN.)	O ₂ CONT. (VOL. %)	% SAT.	HEMATOCRIT % CELLS
Pneumonectomy	10	15.7	95	42	122	15.2	94	40
Lobectomy	20	16.7	95	43	95	16.3	97	45
Miscellaneous	7	17.5	95	40	58	17.5	97	46

OPERATION	NO. OF PATIENTS*	CO ₂ CONT. (VOL. %)	pH	ALVEOLAR CO ₂		TIME AFTER PLEURA OPEN (MIN.)	CO ₂ CONT. (VOL. %)	pH	ALVEOLAR CO ₂	
				TENSION (MM. HG.)	VOL. %				TENSION (MM. HG.)	VOL. %
Pneumonectomy	10	50.0	7.48	35.0	4.9	122	59.6	7.09	98.0	13.7
Lobectomy	20	57.4	7.51	32.8	4.6	95	57.9	7.22	60.6	9.3
Miscellaneous	7	58.1	7.52	32.3	4.5	58	57.4	7.37	44.0	6.2

*Not all items determined on all patients; for details see Tables I to VI.

TABLE VII, B. INCREASE IN FIXED ACID CALCULATED FROM TABLE VII, A

Before Operation

BEFORE	MM PER L HGB.	β	CORRECTION MM PER L	pCO ₂	TOTAL CO ₂ MM PER L	TOTAL CO ₂ - .0301 pCO ₂ [HCO ₃ ⁻]	pH	[HCO ₃ ⁻] CORRECTED TO pH 7.40	ΔA
Pneumonectomy	8.1	27.2	+1.49	35	26.6	25.5	7.48	27.0	—
Lobectomy	8.4	27.9	+2.12	33	25.9	24.9	7.51	27.0	—
Miscellaneous	8.9	29.4	+2.45	32	26.2	25.2	7.52	27.7	—

At Operation's End

NEAR END	MM PER L HGB.	β	CORRECTION MM PER L	pCO ₂	TOTAL CO ₂ MM PER L	TOTAL CO ₂ - .0301 pCO ₂ [HCO ₃ ⁻]	pH	[HCO ₃ ⁻] CORRECTED TO pH 7.40	ΔA
Pneumonectomy	8.9	29.4	-0.35	98	26.9	24.0	7.09	17.7	+9.3
Lobectomy	8.7	28.6	-3.61	67	26.2	24.2	7.22	20.6	+6.4
Miscellaneous	8.9	29.4	-0.62	44	25.9	24.6	7.37	24.0	+3.7

TABLE VIII. RISE IN ALVEOLAR CARBON DIOXIDE TENSION BEFORE PLEURA OPENED

PATIENT	AGE (YR.)	OPERATION	TIME AFTER ANESTHESIA STARTED (MIN.)	CHANGE IN ALVEOLAR CO ₂ TENSION (MM. HG.)
B. W.	49	Pneumonectomy	30	+ 2.1
H. V.	41	Pneumonectomy	40	+16.5
A. A.	40	Lobectomy	40	+ 2.4
K. K.	54	Lobectomy	38	+15.0
F. S.	55	Lobectomy	35	+29.0
E. D.	52	Cyst (mediast.)	55	+ 6.1
R. M.	31	Lobectomy	40	+ 2.2
R. R.	55	Lobectomy	44	+19.3
C. B.	36	Pneumonectomy	60	+40.5
W. B.	27	Lobectomy	35	+13.0
[M. N.]	59	Lobectomy	40	- 4.2]
Average (exclusive of last case)			42	14.6 mm. Hg. (2.0 vol. %)

TABLE IX. PATIENT SUPINE*; VARIOUS OPERATIONS (NONTHORACIC)

PATIENT	SEX	AGE (YR.)	ANES- THESIA TIME (MIN.)	BEFORE OPERATION			AT OPERATION'S END		
				pH	CO ₂ (VOL. %)	CO ₂ TENSION (MM. HG)	pH	CO ₂ (VOL. %)	CO ₂ TENSION (MM. HG)
I. S.	♀	66	255	7.45	63.6	40	7.45	60.3	39
S. T.	♀	66	195	7.45	65.6	41	7.50	57.2	33
G. F.	♀	28	170	7.38	57.3	42	7.35	49.5	38
H. M.	♀	39	95	7.37	54.1	42	7.37	52.9	41
A. G.	♂	66	93	7.40	65.6	48	7.40	58.2	42
J. B.	♂	72	87	7.35	54.1	42	7.35	50.8	40
M. S.	♀	56	120	7.40	61.3	42	7.38	59.0	41
Average				7.40	60.2	42	7.40	55.4	39

*Closed (circle filter) nitrous oxide-oxygen ether anesthesia, size 32 intratracheal tube.

proved to have a mortality rate of their own due to explosion. The unsatisfactory ventilation sometimes produced by these machines is well known. It is widely accepted that respiration carefully assisted by the anesthetist, by bag squeezing just as the patient inspires, gives the most effective type of ventilation. The following two cases, Table X, show how effective this technique is in one case (J.V.) and how unsatisfactory in the other (C.B.). However, the possible effectiveness of assisted respiration needs to be and is being studied further. In current work assistance of the respiration is started as soon as the patient is placed on his side and is continued until the chest is closed. The results of this study will be reported later, when we have had opportunity to observe an adequate number of cases.

The serious nature of the carbon dioxide tensions achieved by the end of long operations through the thorax can be inferred from the abundant evidence on this score found in man not under anesthesia. Possibly the anesthesia may afford some protection. It seems unwise to depend upon that.

Goldstein and DuBois⁵ have shown that a fall of blood pressure immediately follows the cessation of rebreathing of high concentrations of carbon

TABLE X. ARTERIAL BLOOD

PATIENT	SEX	AGE (YR.)	OPERATION	BEFORE OPERATION			TIME AFTER PLEURA OPENED (MIN.)	NEAR END OPERATION			TIME AFTER PLEURA OPENED (MIN.)	AFTER ASSISTED RESPIRATION		
				CO ₂ CONTENT (VOL. %)	pH	ALVEOLAR TENSION (MM. HG)		CO ₂ CONTENT (VOL. %)	pH	ALVEOLAR TENSION (MM. HG)		CO ₂ CONTENT (VOL. %)	pH	ALVEOLAR TENSION (MM. HG)
C. B.	♂	57	Right pneu- monectomy (carcinoma)	62.0	7.55	26.3	180	72.3	7.03	113.8	196	68.5	7.08	90.7
J. V.	♂	84	Left pneu- monectomy (congenital cyst involving entire lung)	64.1	7.47	39.3	72	68.0	7.20	75.0	87	58.4	7.38	43.5

dioxide. Dripps⁷ has discussed the fall of blood pressure which occurs when then subject is taken off cyclopropane. Under this agent he reported that the carbon dioxide tension rises to great heights; a considerable fall of blood pressure sometimes occurs when the cyclopropane is terminated. “. . . the higher the tensions of this gas (carbon dioxide) in the arterial blood the greater is the likelihood of a postanesthetic decrease in blood pressure . . .”

After the end of the surgical procedure and the change of the patient's position to a more favorable one, the pH rises and the carbon dioxide tension is reduced. The stimulant action of the carbon dioxide is withdrawn and the blood pressure falls. As pointed out previously, there is reason to believe that this is a true cause and effect relationship.

The relevance of these findings to the fall of blood pressure often found at the end of thoracic surgical procedures is quite unproved, of course, but a true relationship is probable.

Experience has shown that one can usually “get away” with patients in the position generally used (operated lung uppermost); with only occasionally assisted respiration, what appear to be satisfactory results can be obtained. The disturbing inference remains that the occasional bad results mentioned at the beginning of this paper may in some cases be directly related to the high carbon dioxide tensions reported here.

Possibly surgeons working through the open thorax may decide that they prefer to accept the real hazards of the high carbon dioxide tensions mentioned, tensions that arise as a consequence of common present practices. They may decide that this type of surgery in the supine or prone positions for example is more dangerous than the elevated carbon dioxide tensions. Such a matter is beyond our competence to decide.

In conclusion, reconsideration must be given to what constitutes the truly optimum position for operation in these cases. Equally clearly, more study based on data obtained in man must be given to effective means of assisting respiration during thoracic surgery.

SUMMARY

The blood gases, oxygen and carbon dioxide, the pH and the hematocrit value were measured before anesthesia and at intervals during surgery through the open pleura, and in some instances on the day following operation. Forty-three patients were studied. All of them received nitrous oxide-oxygen ether anesthesia.

Of the two great problems, oxygen supply and carbon dioxide removal, it was found that the techniques employed were adequate to provide oxygen but not to remove carbon dioxide. This gas rises to dangerous tensions with the passage of time. Even though the actual carbon dioxide content does not increase, the rise in tension is effected by a serious fall in pH. Various factors of possible importance in the rise of carbon dioxide tension are considered and discussed. The rise begins shortly after the patient is placed in the lateral position and before the pleura is opened. It continues with increasing rapidity (see Fig. 1).

It is shown (Table IX) that ether anesthesia, the closed anesthesia apparatus, and the endotracheal tube are not responsible, for no such rise occurs when these same factors are present in patients in the supine position. (It can be observed Table IX, that contrary to the statements made in nearly all textbooks of pharmacology, ether does not cause clinical acidosis.) Attention is called to a probable relationship between the high carbon dioxide tensions and the sudden fall of blood pressure with occasional fatality at the end of operation. In conclusion, further consideration should be given to what constitutes the optimum position for surgery in transthoracic procedures and to effective ways of assisting the respiration.

REFERENCES

1. Austin, J. H.: A Note on the Estimation of Carbon Dioxide in Serum in the Presence of Ether by the Van Slyke Method, *J. Biol. Chem.* 61: 345, 1924.
2. Beecher, H. K.: Principles of Anesthesia for Lobectomy and Total Pneumonectomy, *Acta med. Scandinav. (Supp.)* 90: 146, 1938.
3. Beecher, H. K.: II. Some Controversial Matters of Anesthesia for Thoracic Surgery, *J. THORACIC SURG.* 10: 202, 1940.
4. Beecher, H. K., Francis, L., and Anfinsen, C. B.: Acid-base Balance During Ether Anesthesia in Man, *J. Pharmacol. & Exper. Therap.* In press, 1949.
5. Campbell, J. M. H., Douglas, C. G., Haldane, J. S., and Hobson, F. G.: The Response of the Respiratory Center to Carbonic Acid, Oxygen, and Hydrogen Ion Concentration, *J. Physiol.* 46: 301, 1913.
6. Dripps, R. D., and Dumke, P. R.: The Effect of Narcotics on the Balance Between Central and Chemoreceptor Control of Respiration, *J. Pharmacol. & Exper. Therap.* 77: 290, 1943.
7. Dripps, R. D.: The Immediate Decrease in Blood Pressure Seen at the Conclusion of Cyclopropane Anesthesia: "Cyclopropane Shock," *Anesthesiology* 8: 43, 1947.
8. Goldstein, J. D., and Dullois, E. L.: The Effect on the Circulation in Man of Rebreathing Different Concentrations of Carbon Dioxide, *Am. J. Physiol.* 81: 650, 1927.
9. Hastings, A. B., and Sendroy, J., Jr.: The Colorimetric Determination of Blood pH at Body Temperature Without Buffer Standards, *J. Biol. Chem.* 61: 695, 1924.
(See also Peters and Van Slyke Methods [loc. cit.], p. 796.)
10. Peters, J. P., and Van Slyke, D. D.: Quantitative Clinical Chemistry, Methods, Vol. II, ed. 1, Baltimore, 1932, Williams and Wilkins Company, p. 294.
Idem: Interpretations, Vol. I, ed. 4, Baltimore, 1937, Williams and Wilkins, pp. 881, 883.
(See also Van Slyke, D. D., and Sendroy, J., Jr.: Studies of Gas and Electrolyte Equilibrium in Blood, *J. Biol. Chem.* 79: 781, 1928. Line charts for graphic calculations by the Henderson-Hasselbalch equation and for calculating plasma carbon dioxide content from whole blood content.)
11. Pitts, R. F.: In Howell's Textbook of Physiology, ed. 15, Philadelphia, 1946, W. B. Saunders Company, p. 914.
12. Rourke, M. D., and Ernstene, A. C.: A Method for Correcting the Erythrocyte Sedimentation Rate for Variations in the Cell Volume Percentage of Blood, *J. Clin. Investigation* 8: 545, 1930.
13. Shaw, J. L., and Downing, V.: The Determination of Oxygen in Blood in Presence of Ether by a Modification of the Van Slyke-Neill Technique, *J. Biol. Chem.* 109: 403, 1935.
14. Snyder, J. C.: The Cardiac Output and Oxygen Consumption of Nine Surgical Patients Before and After Operation, *J. Clin. Investigation* 17: 571, 1938.
15. Van Slyke, D. D., and Neill, J. M.: The Determination of Gases and Other Solutions by Vacuum Extraction and Manometric Measurement, *J. Biol. Chem.* 61: 523, 1924.

DISCUSSION

DR. JOHN H. GIBBON, JR., Philadelphia.—This presentation interests me very much, and I think Dr. Beecher is to be congratulated on making this very careful study. I only rise to comment about it with the hope that perhaps he will try mechanical respiration and see whether it alters the acidosis which he observed. Since Crafoord, ten years ago, first talked about using a mechanical apparatus to ventilate the lungs in the presence of an open thorax, we have been interested in the method. Dr. Mautz of Cleveland has constructed a simpler apparatus for the same purpose. Last November we obtained Dr. Mautz'

apparatus through his kindness, and we also have the latest model of Crafoord's machine from Stockholm. Since then we have used these two machines on 76 patients; 66 of these had thoracotomies and 10 had upper abdominal operations.

I hope Dr. Beecher will mention some of the difficulties of blood gas determinations in the presence of ether anesthesia. We have made arterial blood gas determinations with open thoracotomies using sodium pentothal curare anesthesia without any ether. The operations were from two to five hours' duration. A needle was inserted in the brachial artery under local anesthesia. A blood sample was taken before the general anesthetic was started. Other samples were taken during, and just at the end of, the operation. With the Mautz machine, we had a significant rise in arterial CO_2 tension in only 1 case out of 5. In 6 of the 9 cases in which we used Crafoord's machine, there was a slight rise, but not to the extent Dr. Beecher reported.

I hope Dr. Beecher will try mechanical respiration and repeat his observations. Unfortunately, we have no arterial blood gas studies without mechanical respiration to compare with the results we have just mentioned.

Finally, I think it is fair to point out, as Dr. Beecher has emphasized, that once an increase in alveolar CO_2 tension occurs it is rather difficult to reduce it. In one case he was successful and in the other not so successful. I think what we all want to do is to avoid getting the increase if we possibly can. I think this is a very interesting work, and I hope the study will be continued.

DR. BEECHER.—I would like to thank Dr. Gibbon for his remarks, and to say that the question he raised about measuring blood gas in the presence of ether is a fairly technical business, and I should like to discuss it outside the meeting as I do not believe it would interest enough of those here. I wondered if in his cases he had taken the blood, where he found a relatively small rise, before the mask was removed. Some of the CO_2 elevations will adjust themselves very rapidly and others take a considerable period of time.

I did not mean to imply that we had a closed mind about any ways of improving this situation. I am very sure, from the fact that these CO_2 tensions begin to rise before the pleura is opened when the respiratory minute volume and rate seem to be within quite reasonable limits, that a normal type of respiration is not the whole answer to correcting this difficulty. I still think it would be desirable for surgeons to consider the possibility of other operating positions for these cases. We anesthetists on our side must find improved ways of assisting respiration in these cases—improvements based upon determinations of arterial oxygen and CO_2 and pH.

BLOOD VOLUME CHANGES IN TUBERCULOUS PATIENTS TREATED BY THORACOPLASTY

FRANK F. ALIBRITTEN, JR., M.D., HERBERT LUSHUTZ, M.D. (BY INVITATION),
BERNARD J. MILLER, M.D. (BY INVITATION), AND JOHN H. GIBBON, JR., M.D.
PHILADELPHIA, PA.

THE patient with pulmonary tuberculosis, when presented for thoracoplasty, frequently has lost² weight, is anemic, and has had prolonged pulmonary suppuration. The circulating blood volume, the total plasma protein, and total hemoglobin are rarely measured although a diminished blood volume in debilitated patients has been noted by Clark,² Gregersen,¹⁰ and Varco.¹⁸ Though it would be suspected that patients with far-advanced pulmonary tuberculosis would fall into this group of patients, staged thoracoplasty is generally well tolerated.

We have found occasional patients who did not tolerate thoracoplasty well, and have been forced by a rising pulse rate or falling blood pressure to terminate the procedure before the desired collapse was obtained. This has occurred despite the fact that the blood lost during the operative procedure was adequately replaced. This occurred more frequently during the second and third stages of thoracoplasty. A reduced blood volume might account for this. To obtain pertinent data, preoperative blood volumes were determined, blood loss at operation was measured, and blood loss between stages of thoracoplasty was determined. The last determination has received little attention, and it occurred to us this additional drain on a depleted blood volume might account for the frequency of shock in the later stages of thoracoplasty.

No reference in the literature to changes in blood volume in patients with pulmonary tuberculosis was found. Blood loss during thoracoplasty and other thoracic procedures has been reported. Stewart and Rourke,¹⁴ using the acid hematin method of blood loss determination of Gatch and Little,⁶ found the blood loss in four thoracoplasties varied from 400 to 900 c.c. Neither the disease of the patient nor the stage or extent of the thoracoplasty was stated. White and Buxton,¹⁷ using the same method, estimated an average 700 c.c. blood loss with each of the three stages of thoracoplasty, but no attempt was made to determine blood lost in the interval between the stages.

Thomas, Adams, and Thornton,¹⁵ in their study of blood volume changes following intrathoracic operations, made no exact measurement of blood loss at the time of operation in most instances. However, in five nontuberculous cases they did determine the blood volume before and after operation using the method of Gibson and Evans⁷ adapted to the photoelectric microcolorimeter by Gibson and Evelyn.⁹ After determining the erythrocyte count, hemoglobin, hematocrit value, plasma protein concentration pre- and postoperatively, and the surgeon's estimate of the blood loss, they came to the conclusion that from

From the Department of Surgical Research, Jefferson Medical College.
Read at the Twenty-ninth Annual Meeting of The American Association for Thoracic Surgery, New Orleans, La., March 29-31, 1949.

a practical standpoint the hematocrit reading was the most valuable index in the determination of blood loss and blood replacement. Ebert, Stead, and Gibson,⁵ in studying the response of normal subjects to acute blood loss, reported that the hematocrit reflected the direction of changes in plasma volume. Stewart and Rourke¹⁴ found no relationship between changes in the hematocrit value, hemoglobin, plasma protein, and the amount of blood lost. They concluded that these determinations should not be used as criteria for fluid replacement. Collier, Crook, and Iob,³ have corroborated their findings.

METHOD

Blood lost at operation was determined by two methods. All sponges were weighed before and immediately after use as described by Wangenstein,^{1, 16} and weights were determined to the nearest 0.1 Gm. The difference in weight was taken to represent the weight of blood lost. Blood volume determinations were made before and after operation using the method of Gibson and Evans⁷ as modified by Gibson and Evelyn⁹ for use with a photoelectric microcolorimeter. The blood was drawn without stasis from a large vein in the arm, usually the median antecubital. The syringe was then removed and the needle left in the vein. A known amount of Evans blue dye T-1824 in a calibrated syringe was immediately injected through this same needle. Blood samples were taken 8, 10, 12, and 14 minutes after injection of the dye. The blood was allowed to clot and then was centrifuged at 2,000 r.p.m. for 15 minutes. Specimens showing hemolysis were discarded. The serum containing the dye was drawn off by pipette, and a little over 1 c.c. was placed in a micro-absorption cell of the plunger type, and light absorption was determined in the Evelyn colorimeter using a 620 m μ filter. Light absorption of the same specimen when using a 540 m μ filter was then determined to correct for unrecognized hemolysis. The galvanometer reading made with the 620 m μ filter was then corrected. The corrected galvanometer readings were then plotted against the time the dye samples were taken, and the galvanometer readings extrapolated to zero time were used to calculate the plasma volume. The blood volume was calculated from the hematocrit and plasma volume. Plasma protein concentration was determined by the copper sulfate method of Phillips and associates.¹³ Total circulating plasma protein concentration was calculated from the plasma volume and protein concentration.

The procedure used in the present series is identical with that described by Miller, Gibbon, and Allbritten¹² in this laboratory. The standard error of estimation as determined by Miller and associates¹² was the same. It was determined by noting the difference between repeat determinations of blood volumes in twenty-five subjects. The repeated determinations were done on each subject within a twenty-four hour period. The differences lay between 0.4 and 7 per cent. The mean experimental difference was 4 per cent. The standard error of estimation was ± 176 c.c. The preoperative blood volume determination was made the day preceding operation. The postoperative blood volume studies were carried out within 30 minutes after completion of the operation. No patient was in shock. Blood loss was calculated by adding the amount of blood given during operation to the preoperative blood volume and subtracting the postoperative blood volume from the sum.

The amount of blood lost between operative procedures was estimated by determining the difference between the postoperative blood volume of any given stage and the preoperative blood volume of the following stage of thoracoplasty and adding the amount of blood transfused during the same interval.

RESULTS

Observations were made on ten patients with pulmonary tuberculosis treated by thoracoplasty. These consisted first, of body weight changes that occurred prior to entering the surgical service; second, the amount of blood lost during

and between the stages of thoracoplasty; third, the amount of blood needed to replace operative and interval loss; and finally the changes that occurred in the hematocrit reading and total circulating plasma protein as a result of each stage of thoracoplasty.

Using normal standards of sex, weight, height, and age³ as a basis for estimation, three of the ten patients studied were of normal or greater than normal weight; seven patients were 8 to 33 pounds underweight. All had far-advanced pulmonary tuberculosis. Five patients had unilateral disease, and five patients had bilateral disease with a contralateral pneumothorax.

Using the chart of Gibson and Evans⁴ to calculate the expected blood volume from the surface area of the patient, all except one patient had a decreased circulating blood volume when admitted to the surgical service. The minimum decrease was 4.5 per cent and maximum decrease 27.5 per cent, and the average was 15.6 per cent below the blood volume expected. Clark and associates² stated that the blood deficit should be calculated on the basis of usual weight prior to illness. If the expected blood volume had been estimated from a surface area calculated from the normal weight for the patient's sex, height, and age,⁴ the minimum decrease would have been 4.7 per cent, the maximum 36.9 per cent, and the average decrease 18.5 per cent.

The blood loss during a first stage thoracoplasty averaged approximately 830 c.c., during a second stage, 480 c.c., and during a third stage, 400 c.c. These sums represent the difference between the preoperative and postoperative blood volume (Table I). When determined by the sponge weight method, the blood loss averaged 850 c.c. for a first stage, 400 c.c. loss for a second stage, and 260 c.c. for a third stage of thoracoplasty. The correlation between the two methods of determining operative blood loss is shown in Fig. 1.

TABLE I. ESTIMATED BLOOD LOSS DURING STAGES OF THORACOPLASTY

STAGES	CASES	BY WEIGHING SPONGES (C.C. BLOOD)			CASES	BY DYE METHOD (C.C. BLOOD)		
		MINI- MUM	MAXI- MUM	AVER- AGE		MINI- MUM	MAXI- MUM	AVER- AGE
First	8	407	1407	855	10	419	1538	832
Second	10	110	990	404	10	95	958	480
Third	5	97	493	262	7	76	858	397

The average total operative blood loss when determined by differences in blood volume for those patients undergoing three stages of thoracoplasty was 1,670 c.c., and 1,430 c.c. when estimated by sponge weight method (Table II). The average operative loss for those patients who had a two-stage, seven-rib thoracoplasty was 1,520 c.c. as determined by the dye T-1824 method, while operative loss estimated by weighing the sponges was 1,220 c.c. (Table III).

The blood loss between the first and second stages of thoracoplasty averaged 430 c.c. and in the interval between the second and third stages this loss averaged 702 c.c. (Table IV). This figure includes the blood lost by one patient (C. K.) by an excessive hemorrhage after the operation. If this figure is eliminated, the

average loss between the second and third stages is 445 c.c. There was no significant blood loss between the third stage and the time of discharge, while the average blood loss between the second stage and the time of discharge in the patients who had only two stages of thoracoplasty was 418 c.c. (Table V).

The operative blood loss was inadequately replaced at the time of operation in four instances by amounts varying from 260 to 460 c.c. Three of these four patients received transfusions of blood upon return to their room. Most patients received interval transfusions to replace loss of blood into the extrafascial space in the interval between operations.

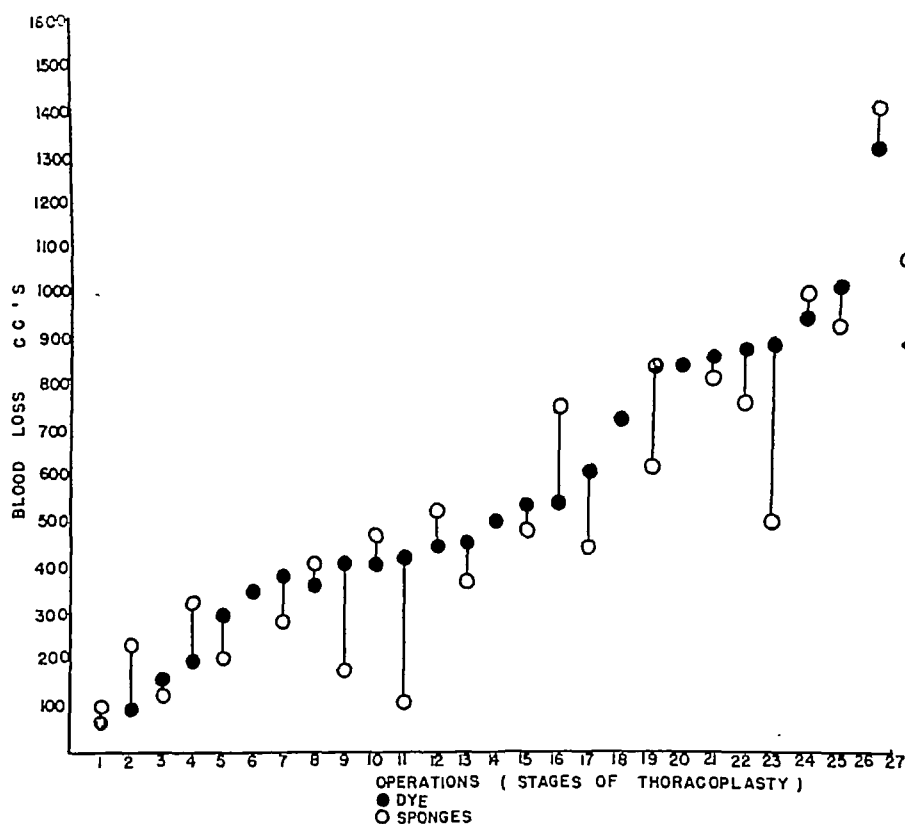


Fig. 1.—Comparison of blood loss as determined by sponge-weighing method and dye (T-1824) method as observed in stages of thoracoplasty.

Hematocrit and total circulating plasma protein concentration showed a trend only suggesting blood deficiency or overreplacement (Figs. 2 and 3). Variations were too great to use these as a basis for accurate estimate of blood lost or blood overreplaced in individual instances.

A miscellaneous group of eleven patients were followed from three months to one year after thoracoplasty. Five patients with arrested disease had gained weight, and the blood volumes had risen to a level nearly equal to the expected blood volume for the surface area. Of these five patients, one had bilateral tuberculosis with contralateral pneumothorax and four had unilateral tubercu-

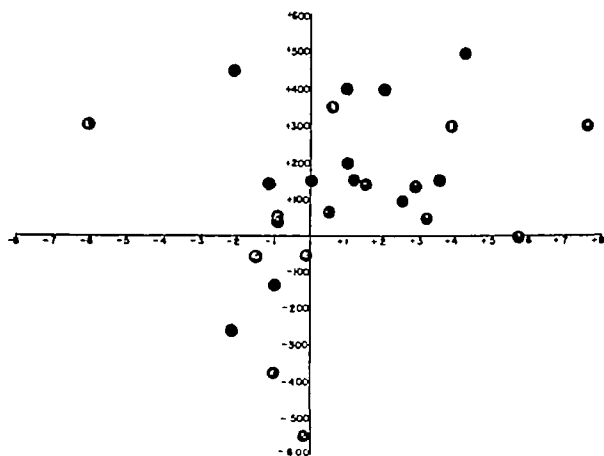


Fig. 2.—Relation of changes in hematocrit to overreplacement and underreplacement of blood at each stage of thoracoplasty. The increase or decrease in hematocrit is the abscissa, and the overreplacement or underreplacement of the blood is the ordinate in the graph.

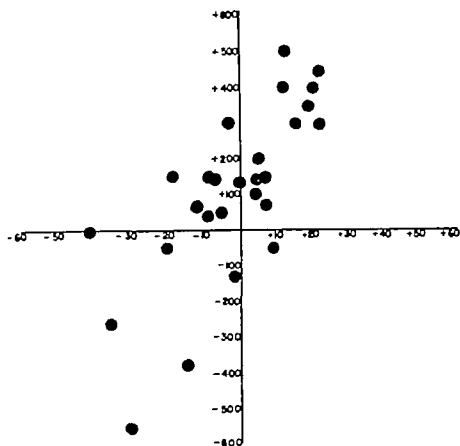


Fig. 3.—The relation of changes in total circulating plasma protein to overreplacement or underreplacement of blood at each stage of thoracoplasty. The increase in grams in the total circulating plasma protein is charted as the abscissa, and the blood replacement in cubic centimeters is charted as the ordinate.

TABLE II. TOTAL OPERATIVE BLOOD LOSS FOR SEVEN RIB THORACOPLASTY COMPLETED IN THREE STAGES

ESTIMATED BY WEIGHING SPONGES			ESTIMATED BY DYE METHOD		
CASES			CASES		
3	Minimum	1156	7	Minimum	1019
	Maximum	1776		Maximum	2672
	Average	1427		Average	1674

TABLE III. TOTAL OPERATIVE BLOOD LOSS FOR SEVEN RIB THORACOPLASTY COMPLETED IN TWO STAGES

ESTIMATED BY WEIGHING SPONGES			ESTIMATED BY DYE METHOD		
CASES					
3	Minimum	967		Minimum	1313
	Maximum	1410		Maximum	1767
	Average	1223		Average	1523

TABLE IV. INTERVAL BLOOD LOSS BETWEEN STAGES

	BETWEEN FIRST AND SECOND STAGES IN 10 CASES (C.C.)	BETWEEN SECOND AND THIRD STAGES IN 7 CASES (C.C.)
Minimum	+14	+254
Maximum	991	2241*
Average	428	702 (445)†

*Includes blood loss of patient (C.K.) who had excessive hemorrhage.

†Excludes blood loss of (C.K.).

TABLE V. INTERVAL BLOOD LOSS BETWEEN LAST OPERATION AND DISCHARGE

	BETWEEN SECOND STAGE AND DISCHARGE IN 3 CASES (C.C.)	BETWEEN THIRD STAGE AND DISCHARGE IN 7 CASES (C.C.)
Minimum	203	+736
Maximum	725	543
Average	418	+10

TABLE VI. CALCULATED TOTAL INTERVAL LOSS

CASES	THREE STAGES	CASES	TWO STAGES
6	Minimum	3	Minimum
	Maximum		Maximum
	Average		Average
	517		771
	1078		1025
	830*		884

*Interval blood loss of patient (C.K.) eliminated because of excessive interval hemorrhage.

TABLE VII. TOTAL BLOOD LOSS DURING THORACOPLASTY

CASES	THREE STAGES	CASES	TWO STAGES
6	Minimum	3	Minimum
	Maximum		Maximum
	Average		Average
	1934		2168
	2984		2792
	2385*		2407

*Interval blood loss of patient (C.K.) eliminated because of excessive interval hemorrhage.

losis. The other six patients still had active pulmonary tuberculosis. These six patients had not gained weight, and the blood volumes had not increased above the preoperative figures.

DISCUSSION

It has been pointed out by several investigators, Clark and associates,² Gregersen,¹⁰ and Varco,¹⁸ that a patient with chronic illness generally has a decreased circulating blood volume. In ten patients with pulmonary tuberculosis, it was observed that although the weight of three of the patients was normal,

or above normal, the blood volume was decreased in all but one, and, when estimated by surface area, averaged 15.6 per cent below the expected figure. Seven of the patients had lost weight, and two of the patients required repeated blood transfusions before thoracoplasty could be started. Clark and associates² stated that correction of blood volume deficits by transfusion has proved to be the most important single factor in the preparation of such patients for surgery. Blood replacement during the operation and in the intervals between the stages of thoracoplasty is equally important. It is during these intervals between the stages that an unrecognized loss of serosanguineous fluid into the operative wound space occurred. The long posterolateral incision, multiple periosteal incisions, and the creation of a large extrafascial space are all conducive to exudation. The space created by the resection of the upper three to five ribs remains until obliterated by subsequent operative procedures. Although it was known that blood and fluid did extravasate into the space, it was thought to be of little consequence. The results of these determinations showed that the amount of blood lost in this manner is significant and that chronically ill patients undergoing a multiple staged operative procedure will be aided by the replacement of the blood lost during that time. Though blood loss at operation was adequately replaced, occasionally thoracoplasty had to be discontinued after giving the anesthetic, for the pulse rate rose and the blood pressure decreased beyond an acceptable point. This occurred more commonly in patients with far-advanced bilateral disease and in the second and third stages of thoracoplasty.

Corroborating the work of Stewart and Rourke¹⁴ and Coller and associates,³ it was found that neither the hematocrit nor the total circulating plasma protein were reliable indices of the need of blood replacement. Griffin and associates,¹¹ also found that the plasma volume and the total circulating plasma albumin varied so widely in normal individuals that they were of no value in estimating dehydration or protein depletion in patients with malnutrition.

Clark and associates² stated that restoration of the blood volume preceded significant recovery of weight during recuperation from debilitating illness. Our results corroborated this statement. Patients with quiescent or arrested disease have shown a return of their blood volumes to a normal range, a weight increase, and a tolerance for mild exercise. In those patients with persistent active disease the blood volume remained low or decreased.

SUMMARY

1. Ten patients with pulmonary tuberculosis had a decreased blood volume upon their admission to the surgical service.
2. Blood lost during the stages of thoracoplasty was measured by the sponge weight method and calculated by differences in circulating blood volumes before and after operation.
3. There is a significant loss of blood between stages of thoracoplasty.
4. Blood replacement is required at the time of operation and during the interval between the stages of thoracoplasty to maintain an optimum blood volume.

5. Neither the hematocrit nor the total circulating plasma protein is an accurate indicator of the amount of blood lost or replaced.

6. An increase in the circulating blood volume of the patients studied paralleled their general clinical improvement.

The technical assistance of Mr. Franz Goldstein is gratefully acknowledged.

REFERENCES

1. Baronofsky, I. D., Treolar, A. E., and Wangenstein, O. H.: Blood Loss in Operations; Statistical Comparison of Losses as Determined by Gravimetric and Colorimetric Methods, *Surgery* 20: 761-769, 1946.
2. Clark, J. H., Nelson, W., Lyons, C., Mayerson, H. S., and Decamp, P.: Chronic Shock: The Problem of Reduced Blood Volume in the Chronically Ill Patient, *Ann. Surg.* 125: 618-646, 1947.
3. Coller, F. A., Crook, C. E., and Iob, V.: Blood Loss in Surgical Operations, *J. A. M. A.* 126: 1-5, 1944.
4. Davenport, C. B.: Body Build and Its Inheritance, Publication 329, Carnegie Institution of Washington, 1923.
5. Ebert, R. V., Stead, E. A., Jr., and Gibson, J. G., Jr.: Response of Normal Subjects to Acute Blood Loss, *Arch. Int. Med.* 68: 578-590, 1941.
6. Gatch, W. D., and Little, W. D.: Amount of Blood Lost During Some of the More Common Operations, *J. A. M. A.* 83: 1075-1076, 1924.
7. Gibson, J. G., Jr., and Evans, W. A., Jr.: Clinical Studies of Blood Volume; Clinical Application of a Method Employing the Azo Dye "Evans Blue" and Spectrophotometer, *J. Clin. Investigation* 16: 301-316, 1937.
8. Gibson, J. G., Jr., and Evans, W. A., Jr.: Clinical Studies of Blood Volume, II, The Relation of Plasma and Total Blood Volume to Venous Pressure, Blood Velocity Rate, Physical Measurements, Age and Sex in Ninety Normal Humans, *J. Clin. Investigation* 16: 317-328, 1937.
9. Gibson, J. G., Jr., and Evelyn, K. A.: Clinical Studies of Blood Volume, IV, Adaptation of the Method to the Photoelectric Microcolorimeter, *J. Clin. Investigation* 17: 153-158, 1938.
10. Gregersen, M. I.: A Practical Method for the Determination of Blood Volume With the Dye T-1824, *J. Lab. & Clin. Med.* 29: 1266-1286, 1944.
11. Griffin, G. E., Abbott, W. E., Pride, M. P., Muntwyler, E., Mautz, F. R., and Griffith, L.: Plasma Volume "Available (Thiocyanate) Volume," and Total Circulating Plasma Proteins in Normal Adults, *Ann. Surg.* 121: 352-360, 1945.
12. Miller, B. J., Gibbon, J. H., Jr., and Allbritten, F. F., Jr.: Blood Volume and Extracellular Fluid Changes During Thoracic Operations, *J. THORACIC SURG.* 18: 605, 1949.
13. Phillips, R. A., Van Slyke, D. D., Dole, V. P., Emerson, K. M., Jr., Hamilton, P. B., and Archibald R. M.: Copper Sulfate Method for Measuring Specific Gravities of Whole Blood and Plasma, *Bull. U. S. Army M. Dept.* 71: 66-83, 1943.
14. Stewart, J. D., and Rourke, G. M.: Changes in the Blood and Interstitial Fluid Resulting From Surgical Operation and Ether Anesthesia, *J. Clin. Investigation* 17: 413-416, 1938.
15. Thomas, L. B., Adams, W. E., and Thornton, T. F., Jr.: Alterations of the Blood Following Intrathoracic Operations, *Surgery* 20: 38-55, 1946.
16. Wangenstein, O. H.: The Controlled Administration of Fluid to Surgical Patients, *Minnesota Med.* 25: 783-789, 1942.
17. White, M. L., and Buxton, R. W.: Blood Loss in Thoracic Operations, *J. THORACIC SURG.* 12: 198-202, 1942.
18. Varco, R. L.: Preoperative Dietary Management for Surgical Patients With Special Reference to Lesions of Stomach and Duodenum, *Surgery* 19: 303-378, 1946.

DISCUSSION

DR. D. H. WATERMAN, Knoxville, Tenn.—To my mind this is a highly significant paper, and one I do not wish to let go without discussion. In spite of the dramatic things that are occurring in thoracic surgery, we must not lose sight of the fact that thoracoplasty is the common operation that we are doing day in and day out, and that the excellent data Dr. Allbritten and his associates have presented certainly point the way to more adequate management of this large group of patients.

I trust there are no longer thoracic surgeons who pride themselves on never using transfusion in thoracoplasty; some years ago that was a not uncommon attitude. I am sure that all of us now routinely use transfusion in every case of thoracoplasty, and that many of us are coming to the point of giving a transfusion in the interval between stages as well.

I might mention that in our own series we have routinely, in the last 100 cases of thoracoplasty, given two pints of blood at the first stage and one at each of the succeeding stages, with quite a large percentage of patients now receiving another 500 c.c. transfusion in the interval. I am happy to see that these data have confirmed the clinical impressions we have had that there must be a further blood loss during this interval. I wish again to congratulate Dr. Allbritten on this study.

DR. EMILE HOLMAN, San Francisco.—I too wish to congratulate the speaker on these significant findings. I thought you might be interested in a little indirect evidence of the decreased blood volume in these patients. (Slides.)

You are all acquainted with the change in size of the heart that corresponds with blood loss imposed on a dog, for example; this is the normal heart size. Remove 300 c.c. of blood and we have a heart that is correspondingly smaller; remove 300 c.c. more blood and the heart becomes even smaller. Restore the blood and it is a little larger than the original heart, because in the meantime the circulating blood has been increased by accretions from the blood reservoirs, such as the spleen and body tissues. No doubt you have all noted the very small heart (slides) in the presence of pulmonary tuberculosis, which we have been ascribing to a decreased blood volume. About ten years ago a young Chinese doctor, Chang, studied the blood volume in tuberculous patients in our clinic. Unfortunately, probably due to the limitations of the method used, they all turned out normal. The studies this morning indicate that our thesis was sound, and that the small heart noted in tuberculous patients may be considered indirect evidence of a decreased blood volume in these patients.

DR. ADRIAN LAMBERT, New York.—I have been interested in these cases that come to surgery for tuberculosis, especially in patients with tuberculous empyemas who have not been able to withstand surgery. In 1944, we had occasion to do some isolated blood volume studies and found, as Dr. Allbritten did, that their blood volumes were low. We, therefore, went about looking for blood for them, which was not available; we appealed to the Red Cross, and they gave us some red blood cells which we mixed with 10 per cent corn syrup according to the method of Thalheimer, as published by Thalheimer and Stephens. This allowed us to keep the blood viable and useful for more than thirty days, and we gave four transfusions a week for four weeks to a series of approximately ten patients on our wards who were debilitated, with chronic draining wounds. These patients, after this treatment, looked as if they had been to Florida; they were florid in complexion and underwent certain changes which were interesting. They began to gain weight, their appetites increased, and the character of their wounds changed. The granulations became pink and whereas, before, the type of suppuration and purulent material was profuse and thick, it dried up and the secretions became quite watery. We used that as a preoperative measure and thought it enabled them to withstand surgery which they were not otherwise able to do.

DR. ALLBRITTEN, JR.—I wish to thank the discussors. We are not aware of any previous determinations of circulating blood volume in patients with pulmonary tuberculosis or the amount of blood lost in the intervals between stages of thoracoplasty.

Though patients generally receive blood transfusions during thoracoplasty there is a tendency to underestimate the requirements for adequate replacement unless the approximate figures as based upon actual measurement are known.

PULMONARY CIRCULATION AND ALVEOLAR VENTILATION— PERFUSION RELATIONSHIPS AFTER PNEUMONECTOMY

A. COURNAND, M.D., R. L. RILEY, M.D. (BY INVITATION), A. HIMMELSTEIN, M.D.
(BY INVITATION), AND R. AUSTRIAN, M.D. (BY INVITATION)
NEW YORK, N. Y.

THE resection of one lung necessitates readjustments in the mechanics of the chest bellows and a redistribution of blood flow and ventilation through the remaining lung. Several problems concerning the manner in which these adaptive processes take place are of interest to the thoracic surgeon, the physiologist, and the pathologist. In three previous papers, physiologic studies have been reported on the effect of pneumonectomy in a group of 4 young children followed up to adult age^{1, 2} and in a group of adults followed for a short period of time.³ The techniques available when these reports were made included the measurements of (a) lung volumes, (b) maximum breathing capacity, (c) distribution of tidal air throughout the lung, (d) ventilation, and (e) arterial blood oxygen saturation under varying states of activity. The following tentative conclusions were reached on the basis of the data obtained with these techniques: (1) A single lung, provided it is maintained within its normal confines naturally or following a thoracoplasty, is capable of a high degree of efficiency in both the ventilatory and the respiratory aspects of pulmonary function; (2) pulmonary distention, defined as a greater than normal size of the remaining lung in forced expiration, is not functionally desirable in young and adult subjects as it limits the ventilatory capacity of the chest bellows and causes a reduction in the arterial oxygen saturation during a severe type of exercise; and (3) fixation of the mediastinal contents in their normal position, following the use of pneumothorax, oleothorax, or thoracoplasty corrects the latter disturbances. Birath,⁴ who was concerned chiefly with the ventilatory aspects of the adaptation, has since confirmed that a distended lung loses some of its efficiency in distributing tidal air. Since these studies were undertaken two new methods have been introduced in clinical investigation; that is, cardiac catheterization⁴ and the measurement of partial pressures of carbon dioxide and oxygen in the arterial blood,^{5, 6} which methods might give interesting information regarding the effect of pneumonectomy upon the pulmonary ventilation and circulation. With the cardiac catheterization technique, it is possible to study the variation in pulmonary blood flow and pulmonary arterial pressure^{7, 8, 9} at rest and during a steady state of moderate exercise. By combining the data obtained from the measurement of partial pressures of carbon dioxide and oxygen in arterial blood with some of the data obtained by catheterization of the heart the relationships existing between alveolar ventilation and perfusion of pulmonary capillaries may be

From the Departments of Medicine and Surgery, Columbia University College of Physicians and Surgeons, and the Chest Service (Columbia University Division), Bellevue Hospital. This work was supported by a grant from the Commonwealth Fund.

Read at the Twenty-ninth Annual Meeting of The American Association for Thoracic Surgery, New Orleans, La., March 29-31, 1949.

closely investigated.¹⁰ The measurement of partial pressures of oxygen in the arterial blood is a more sensitive index of the state of oxygenation of the arterial blood than the measurement of the oxygen saturation, because of the characteristics of the oxyhemoglobin dissociation curve as it approaches full oxygen saturation. The studies to be reported in this paper are based upon analyses of the data obtained with both these methods and also with the methods previously used. The 16 cases which were investigated offer a great diversity with regard to the nature of the pathologic process prior to pulmonary resection, the time interval after pneumonectomy, the age of the subject, and the presence or absence of a thoracoplasty.

MATERIAL FOR STUDY

The 16 cases which form the basis of this report may be divided into two groups. A first group (Group I) of 7 patients was studied within the first five months following pneumonectomy, and a second group (Group II) of 9 patients was studied from 25 to 131 months after pneumonectomy.

In Tables I and II are summarized the physical characteristics and the essential clinical data at the time of the latest study.

TABLE I. PHYSICAL CHARACTERISTICS AND CLINICAL DATA IN A GROUP OF 7 CASES STUDIED WITHIN A FEW MONTHS AFTER PNEUMONECTOMY (GROUP I)

SUBJECT	AGE (YR.)	SEX	WEIGHT (KG.)	HEIGHT (CM.)	BODY SURFACE AREA (SQ. M.)	DIAGNOSIS	DATE OF RESECTION	DATE OF STUDIES
G. H.	48	M	65	177	1.81	Bronchiectasis, left pneumonectomy, no thoracoplasty	4/ 9/48	5/12/48
V. Ab.	35	M	72	171	1.83	Bronchiectasis, bilateral pulmonary emphysema; right pneumonectomy, no thoracoplasty	6/27/47	6/12/47 10/14/47
A. Am.	24	M	75	169	1.86	Bronchiectasis, left pneumonectomy, thoracoplasty, no scoliosis	10/20/47	10/ 2/47 1/22/48
C. Ro.	22	F	50	150	1.43	Tubercular stenosis of left bronchus, left pneumonectomy, thoracoplasty no scoliosis	5/ 7/47	6/17/47
J. Ra.	15	M	54	164	1.59	Tubercular stenosis of right bronchus, right pneumonectomy, thoracoplasty, no scoliosis	5/18/48	11/10/48
C. McL.	34	M	44	166	1.46	Bronchiectasis, right pneumonectomy, thoracoplasty, scoliosis	8/ 0/47	1/20/48
J. Rut.	57	M	54	157	1.53	Bronchiectasis, lung abscess, left pneumonectomy, thoracoplasty, scoliosis	6/28/48	6/ 2/48 9/27/48

TABLE II. PHYSICAL CHARACTERISTICS AND CLINICAL DATA IN A GROUP OF 9 CASES STUDIED SEVERAL YEARS AFTER PNEUMONECTOMY (GROUP II)

SUBJECT	AGE (YR.)	SEX	WEIGHT (KG.)	HEIGHT (CM.)	BODY SURFACE AREA (SQ. M.)	DIAGNOSIS	DATE OF RESECTION	DATE OF STUDIES
H. W.*	17	F	59	165	1.65	Bronchiectasis, left pneumonectomy, no thoracoplasty	10/ 5/37	11/10/48 2/15/49
R. G.*	21	M	63	177	1.78	Bronchiectasis, left pneumonectomy, no thoracoplasty	2/18/38	12/19/47
S. R.	41	M	80	173	1.93	Lung abscess (foreign body), right pneumonectomy, no thoracoplasty	11/15/45	12/17/47
J. Lo.*	19	M	66	171	1.78	Bronchiectasis, left pneumonectomy, no thoracoplasty; pulmonary distention	11/10/39	9/21/48
J. Rus.	61‡	M	46	158	1.44	Squamous-cell bronchial carcinoma, right pneumonectomy, no thoracoplasty, nonobstructive pulmonary emphysema	2/27/46	4/ 1/46 5/ 5/48
V. Wat.	72‡	M	65	175	1.79	Oat-cell bronchial carcinoma, left pneumonectomy, no thoracoplasty, nonobstructive pulmonary emphysema, systemic arterial hypertension	8/ 2/41	12/12/41 1/26/44 9/18/44 9/ 7/48
D. M.†	66	M	62	179	1.79	Transitional cell bronchial carcinoma, bilateral obstructive emphysema, arteriosclerotic heart disease, left pneumonectomy, no thoracoplasty	11/23/40	12/15/41 2/23/44
E. Re.†	39‡	M	53	175	1.64	Bronchiectasis, left pneumonectomy, thoracoplasty, no scoliosis, bronchiectasis right middle lobe, slight pulmonary distention	7/18/41	3/18/48
F. I.†	53‡	M	69	178	1.86	Tubercular stenosis of right bronchus, right pneumonectomy, thoracoplasty, slight pulmonary distention, mild systemic arterial hypertension	5/19/41	8/10/42 12/13/48

*Cases reported in J. THORACIC SURG. 16: 30, 1947, under same initials.

†Cases reported in Ann. Surg. 116: 532, 1942, under same initials.

‡Age at time of latest study.

METHODS

1. The following measurements were made routinely in the 16 cases and were usually repeated at intervals in the 9 cases of the second group: (a) Lung volume and maximum breathing capacity; (b) pulmonary emptying rate or index of intrapulmonary mixing; (c) ventilation and gas exchange at rest, during a standard exercise test of one minute's duration, and during the first five minutes of recovery following the exercise. The techniques used in these routine studies have been described in detail recently.¹¹ In the same report may be found standard values for normal individuals, which vary with age, sex, and body size.

2. The study of the relationships between alveolar ventilation and alveolar perfusion was based on techniques recently developed, and on a physiologic interpretation of the data obtained by these techniques. The techniques consist of: (a) The collection of blood samples from the brachial artery through an indwelling needle and the determination in these samples of the carbon dioxide ($p\text{CO}_2$) and oxygen ($p\text{O}_2$) tensions by the bubble technique developed by Riley and associates;³ (b) the simultaneous collection of mixed venous blood samples using the technique of cardiac catheterization and the determination of O_2 content and the O_2 saturation in these samples; (c) the simultaneous measurement of the ventilation and of the CO_2 output and O_2 intake from which the respiratory quotient may be calculated. These measurements are made at rest, under basal conditions and during the steady state attained after ten to twelve minutes of a moderate exercise with the subject breathing room air. The exercise was of two types: a leg exercise with the subject in the recumbent position, flat on his back, and/or a bicycle exercise.

On the basis of physiologic concepts developed by Lilienthal and co-workers⁴ and more recently expanded by Riley and Cournand,¹⁰ the data obtained by these techniques may be used to analyze the ventilation-perfusion relationships in the lungs starting with the concept of "ideal" alveolar oxygen and carbon dioxide tensions. These ideal tensions are those which the alveolar air and the blood leaving the pulmonary capillaries would have if there were homogeneous ventilation and perfusion of the alveoli throughout the lungs and if the gas tensions in the alveolar air and in the capillary blood reached perfect equilibrium. The "ideal" alveolo-arterial $p\text{O}_2$ gradient results chiefly from the fact that all alveoli do not contain "ideal" alveolar air because the ventilation-perfusion relationships vary.

To facilitate the quantitative analysis of ventilation-perfusion relationships, all pulmonary alveoli may be considered as if they fell into one of three categories: (1) "ideal" alveoli with "ideal" ventilation-perfusion relationships ("ideal" $p\text{O}_2$ and $p\text{CO}_2$); (2) alveoli which are perfused but not ventilated (mixed venous blood partial pressures); and (3) alveoli which are ventilated but not perfused (inspired air partial pressures). If total pulmonary blood flow (cardiac output) is known, it is then possible to calculate the effective pulmonary blood flow, which is the portion of total pulmonary blood flow perfusing "ideal" alveoli. The ratio of effective pulmonary blood flow to total pulmonary blood flow in the absence of a true shunt provides a quantitative estimate of the

physiologic importance of alveoli which are well perfused but poorly ventilated. If total ventilation is known, it is possible to calculate the effective alveolar ventilation, which is that portion of total ventilation distributed to "ideal" alveoli. The ratio of effective alveolar ventilation to total ventilation provides a quantitative estimate of the physiologic importance of alveoli which are well ventilated but poorly perfused.

A schematic representation of these relationships is given in Fig. 1, in which ventilation is considered as a continuous process.

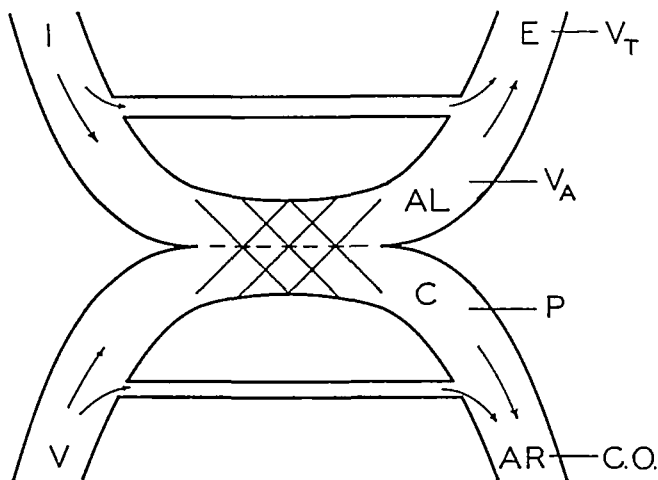


Fig. 1.—Schematic representation of ventilation, perfusion, and gas exchange.¹⁰

The symbols represent

a. The composition of respiratory gas (partial pressure or content): in *I*, inspired air; *E*, expired air; *V*, mixed venous blood; *Ar*, arterial blood; *Al*, "ideal" alveolar air; *C*, blood leaving "ideal" alveoli.

b. The rate of flow in liters per minute: *V_A* effective ventilation; *P*, effective pulmonary blood flow; *V_T*, total ventilation; *C.O.*, total pulmonary blood flow.

The volume of gas, with the composition of inspired air, bypassing the "ideal" alveoli, is influenced by the anatomic size of the bronchial tree, the gaseous diffusion between terminal bronchioles and alveoli, and the proportion of poorly perfused or nonperfused alveoli.

The volume of blood with the composition of mixed venous blood by-passing the "ideal" capillaries is influenced by the proportion of poorly ventilated or nonventilated alveoli.

In normal individuals effective pulmonary blood flow constitutes at least 94 per cent of total pulmonary blood flow and the effective alveolar ventilation constitutes at least 70 per cent of total ventilation.

Deviations from these normal percentage figures, in the absence of technical errors, are of three types, the description and interpretation of which follows:

1. A reduction of both effective pulmonary blood flow and effective alveolar ventilation below the normal percentage indicates abnormalities of distribution of ventilation and perfusion throughout the lungs, the extent of which is determined by the calculated percentage.
2. A reduction of the effective pulmonary blood flow below the normal percentage, combined with an effective alveolar ventilation within the normal percentage range, indicates either passage of blood through totally unventilated portions of the lungs (true shunt) or circulation through poorly ventilated alveoli.

3. A reduction of the effective alveolar ventilation below the normal percentage combined with an effective pulmonary blood flow within the normal percentage range indicates restriction of the circulation through well-ventilated parts of the lungs.

The following formulas were used in these calculations:

$$1. \text{ "Ideal" alv. } pO_2 = \text{Insp. } pO_2 - \frac{\text{Art. } pCO_2}{R.Q.}$$

where

"Ideal" alv. pO_2 = "Ideal" alveolar oxygen tension in mm. Hg

Insp. pO_2 = partial pressure of oxygen in inspired air in mm. Hg corrected for difference in volume between inspired air and alveolar air

Art. pCO_2 = Arterial blood carbon dioxide partial pressure in mm. Hg

R.Q. = Respiratory quotient

$$2. E.P.B.F. = T.P.B.F. \times \left[1 - \frac{X - AR}{X - MVB} \right]$$

where

E.P.B.F. = Effective pulmonary blood flow in liters per minute per m^2 B.S.

T.P.B.F. = Total pulmonary blood flow in liters per minute per m^2 B.S.

X = "Ideal" capillary blood O_2 saturation per cent

AR = Arterial blood oxygen saturation per cent

MVB = Mixed venous blood oxygen saturation per cent

$$3. \text{ Alv. Vent.} = \text{Tot. Vent.} \times \left[1 - \frac{\text{Art. } pCO_2 - \text{Exp. } pCO_2}{\text{Art. } pCO_2} \right]$$

where

Alv. Vent. = Alveolar ventilation in liters per minute per m^2 B.S.

Tot. Vent. = Total ventilation in liters per minute per m^2 B.S. corrected for the effect of apparatus dead space

Art. pCO_2 = Arterial carbon dioxide partial pressure in mm. Hg

Exp. pCO_2 = Expired carbon dioxide partial pressure in mm. Hg

Studies were also performed with the subjects at rest, breathing a mixture of approximately 12 per cent oxygen in nitrogen. Contrary to the situation which obtains during breathing of room air, nearly perfect equilibrium between alveolar pO_2 and the pO_2 of the blood leaving the alveolar capillaries is not reached during low oxygen breathing. Under these circumstances the alveolo-arterial pO_2 gradient is related chiefly to the diffusion characteristics of the tissue-fluid barrier (the "pulmonary membrane") between the blood and gas phases. In normal individuals this gradient is below 12 mm. Hg. From this gradient the diffusion constant for the lung as a whole can be estimated.

RESULTS

The results in the two groups will be discussed separately.

Group I

The data obtained from the 7 patients of this group are tabulated in Tables III, IV, V, VI, VII, XII, XIV, and XV, and may be compared in these tables with normal or predicted values. Measurements were made only once between

TABLE III. LUNG VOLUMES AND MAXIMUM BREATHING CAPACITY IN 7 CASES STUDIED WITHIN A FEW MONTHS AFTER PNEUMONECTOMY (GROUP I)

SUBJECT	TIME AFTER OPERATION (MO.)	VITAL CAPACITY (IN C.C.)		RESIDUAL AIR (IN C.C.)		TOTAL CAPACITY (IN C.C.)		RESIDUAL AIR TOTAL CAPACITY		MAXIMUM BREATHING CAPACITY (LITERS)	
		PREDICTED*	OBSERVED	PREDICTED*	OBSERVED	PREDICTED*	OBSERVED	PREDICTED	OBSERVED	PREDICTED†	OBSERVED
G. H.	1	2160	1905	660	785	2820	2690	23	28	110	92
V. Ab.	Before 4	4050	1360	1230	2410	5280	3770	23	64	129	41
		1823	1867	557	1706	2380	3573	23	48	129	40
A. Am.	Before 3	4200	3987	1050	1753	5250	5740	20	31	147	103
		2310	2565	578	576	2888	3141	20	18	147	108
C. Ro.	1½	1611	1636	403	470	2014	2106	20	22	86	73
J. Ra.	5	1915	1734	480	442	2395	2176	20	20	115	84
C. McL.	5	1776	1142	544	802	2320	1944	23	41	100	38
J. Rut.	Before 3	3810	2554	1475	1184	4785	3738	31	32	63	47
		1820	1701	810	1258	2630	2959	31	43	63	31
Average of 7 cases after pneumonectomy	3	1916	1793	576	863	2492	2656	23	31	107	67

*Predicted volume of a homologous single normal lung.

†Predicted maximum breathing capacity for a normal subject with intact chest and lungs and of the same age, sex and size.

TABLE IV. (A) INDEX OF INTRAPULMONARY MIXING; (B) VENTILATION AND BREATHING RESERVE AT REST, DURING A ONE-MINUTE STANDARD EXERCISE, AND DURING THE FIRST MINUTE OF RECOVERY IN 7 CASES STUDIED WITHIN A FEW MONTHS AFTER PNEUMONECTOMY (GROUP I)

SUBJECT	TIME AFTER OPERATION (MO.)	INDEX OF INTRA-PULMONARY MIXING N ₂ %	BASAL		VENTILATION (IN LITERS PER MIN. PER M ² D.S.)				BREATHING RESERVE (IN LITERS PER MIN.)			
					EXERCISE		RECOVERY		EXERCISE		RECOVERY	
			PREDICTED	OBSERVED								
G. H.	1	.82	3.1	4.1	10.0	17.0	13.4	10.8	92	62	80	43
V. Ah.	Before	-	-	-	-	-	-	-	-	-	-	-
	4	2.3	3.1	4.0	10.0	9.7	13.4	9.9	106	23	93	14
A. Am.	Before	1.90	3.0	4.3	-	-	-	-	-	-	-	-
	8	1.21	3.6	5.7	11.0	14.2	12.5	13.9	117	81	107	60
C. Ro.	1 1/2	.73	3.2	4.0	9.0	17.8	10.9	12.1	73	48	66	45
J. Ra.	5	.95	3.0	3.5	11.0	10.8	12.5	10.8	98	67	80	62
C. McL.	5	1.03	3.6	5.4	11.0	12.5	12.5	12.6	84	18	77	22
J. Rut.	Before	1.09	3.9	5.9	11.2	14.5	14.5	21.4	72	63	63	47
	3	1.37	3.9	5.2	11.2	17.0	14.5	22.0	70	43	61	31
Average of 7 cases after pneumonec-tomy		1.20	3.4	4.6	10.4	14.3	12.8	14.5	91	49	81	39

TABLE III. LUNG VOLUMES AND MAXIMUM BREATHING CAPACITY IN 7 CASES STUDIED WITHIN A FEW MONTHS AFTER PNEUMONECTOMY (GROUP I)

SUBJECT	TIME AFTER OPERATION (MO.)	VITAL CAPACITY (IN C.C.)		RESIDUAL AIR (IN C.C.)		TOTAL CAPACITY (IN C.C.)		RESIDUAL AIR TOTAL CAPACITY $\times 100$		MAXIMUM BREATHING CAPACITY (LITERS)	
		PREDICTED*	OBSERVED	PREDICTED*	OBSERVED	PREDICTED*	OBSERVED	PREDICTED	OBSERVED	PREDICTED†	OBSERVED
G. H.	1	2160	1905	660	785	2820	2690	23	28	110	92
V. Ab.	Before 4	4050 1823	1360 1867	1230 557	2410 1706	5280 2380	3770 3573	23	64	129	41
A. Am.	Before 3	4200 2310	3987 2565	1050 578	1753 576	5250 2888	5740 3141	20	31	147	103
C. Ro.	1½	1611	1636	403	470	2014	2106	20	22	86	73
J. Ra.	5	1915	1734	480	442	2395	2176	20	20	115	84
C. McL.	5	1776	1142	544	802	2320	1944	23	41	100	38
J. Rut.	Before 3	3310 1820	2554 1701	1475 810	1184 1258	4785 2630	3738 2959	31	32	63	47
Average of 7 cases after pneumonectomy	3	1916	1793	576	863	2492	2656	23	31	107	67

*Predicted volume of a homologous single normal lung.

†Predicted maximum breathing capacity for a normal subject with intact chest and lungs and of the same age, sex and size.

TABLE VI. CO₂ AND O₂ PRESSURE GRADIENTS BETWEEN AMBIENT AIR, ALVEOLAR AIR, AND ARTERIAL BLOOD IN 7 CASES STUDIED WITHIN A FEW MONTHS AFTER PNEUMONECTOMY

SUBJECT	TIME AFTER OPERATION (MO.)	STATE	O ₂ TENSIONS (pO ₂) (IN MM. HG)				PO ₂ GRADIENTS (IN MM. HG)		CO ₂ TENSIONS (pCO ₂) (IN MM. HG)		TIDAL AIR (IN C.C.)	RESPIRA- TORY QUOTIENT
			INSPIRED AIR (I)	ALVEOLAR AIR (AL)	ARTERIAL BLOOD (AR)	I-AL	AL-AR	EXPIRED AIR	ARTERIAL BLOOD			
Control Normals		Rest (R. A.)	152	104	94	48	10	20	30	605	.81	
		Exer. (B)	151	109	93	42	14	33	38	1064	.90	
		Rest (Low O ₂)	84	42	33	42	9	23	38	721	.80	
G. H.	1	Rest (R. A.)*	151	95	79	50	16	22	42	474	.75	
		Rest (Low O ₂)	80	58	40	31	12	14	34	538	1.06	
V. Ab.	4	Rest (R. A.)	151	90	80	55	16	21	43	387	.70	
A. Am.	Before	Rest (R. A.)	153	105	85	48	20	23	36	400	.76	
		Exer. (B)	152	105	85	47	20	31	43	1507	.92	
	3	Rest (R. A.)	148	97	83	51	14	21	30	820	.77	
		Exer. (B)	147	98	82	49	16	23	44	1445	.90	
		Rest (Low O ₂)	84	49	44	35	5	22	34	804	.98	
C. Ro.	1½	Rest (R. A.)*	152	104	100	48	4	22	30	338	.81	
		Exer. (B)*	151	118	106	45	12	24	35	558	1.07	
		Rest (Low O ₂)	71	47	37	24	10	15	31	323	1.98	
J. Ru.	5	Rest (R. A.)	153	104	93	49	11	27	30	631	.70	
		Rest (Low O ₂)	91	47	38	44	9	24	38	580	.87	
C. McL.	5	Rest (R. A.)	153	106	84	47	22	10	36	470	.77	
		Exer. (B)	152	113	95	39	18	23	35	673	.89	
		Rest (Low O ₂)	87	49	36	38	13	18	33	450	.88	
J. Rut.	Before	Rest (R. A.)	149	103	85	46	18	15	40	485	.87	
		Rest (Low O ₂)	89	45	40	44	5	15	29	445	.80	
	3	Rest (R. A.)	151	113	94	38	19	17	33	370	.86	
		Rest (Low O ₂)	90	51	44	39	7	15	32	350	.83	

*Respiratory gas tensions studied simultaneously with blood flow measurements by cardiac catheterization technique.

Rest (R.A.), Resting state, breathing room air.

Rest (Low O₂), Rest state, breathing a mixture of 12 per cent oxygen in nitrogen.

Exer. (B), Bicycle exercise with steady state.

For averages see Table XII.

TABLE VII. EFFECTIVE ALVEOLAR VENTILATION AND EFFECTIVE PULMONARY BLOOD FLOW IN 7 CASES STUDIED WITHIN A FEW MONTHS AFTER PNEUMONECTOMY

SUBJECT	STATE	ALV. ARTERIAL PO ₂ GRADIENT (MM. HG)	PULMONARY BLOOD FLOW		RATIO $\frac{B}{A}$	PULMONARY VENTILATION		RATIO $\frac{D}{C}$
			TOTAL (A) (LITERS PER MIN. PER M ² B.S.)	EFFECTIVE (B) (LITERS PER MIN. PER M ² B.S.)		TOTAL (C) (LITERS PER MIN. PER M ² B.S.)	EFFECTIVE ALV. (D) (LITERS PER MIN. PER M ² B.S.)	
Normal	Rest	>12	3.10	2.95	>94	3.82	2.97	>70
G. H.	Rest	16	4.68	4.26	91	4.10	2.66	65
V. Ab.	Rest	16	-	-	91.2*	4.08	2.74	67
A. Am.	Rest	14	-	-	92.9*	5.05	3.50	62
C. Ro.	Rest	4	3.21	3.15	98	4.43	3.28	74
	Exercise	12	4.37	4.30	98.5	12.30	9.80	79
J. Ra.	Rest	11	-	-	95.1*	3.54	2.52	71
C. McL.	Rest	22	-	-	92.6*	5.37	3.60	67
J. Rut.	Rest	19	-	-	92.8*	5.21	3.54	68
Average of 7 cases at rest		15	-	-	93.4	4.63	3.12	68
Average of 2 cases at rest in which MVB was sampled		10	4.15	3.56	94.5	4.27	2.97	70

*In cases where the mixed venous blood oxygen saturation was not determined directly, the ratio of effective to total blood flow was calculated assuming a mixed venous blood saturation 25 per cent lower than the determined arterial blood saturation on the basis of the following formula:

$$\frac{\text{Effective blood flow}}{\text{Total blood flow}} \times 100 = \frac{\text{Art. blood O}_2 \text{ sat. \%} - \text{mixed venous blood O}_2 \text{ sat. \%}}{\text{Total capillary O}_2 \text{ sat. \%} - \text{mixed venous blood O}_2 \text{ sat. \%}} \times 100$$

arterial blood during room air breathing was slightly below the accepted normal value and the alveolo-arterial pO₂ gradient was slightly elevated. Using these figures for the calculation of effective pulmonary blood flow and of effective alveolar ventilation expressed in per cent of total blood flow and of total ventilation, it was found that both were slightly below normal standard values, respectively 93.6 and 68 per cent.

8. The average alveolo-arterial pO₂ gradient while breathing a low oxygen mixture was within the normal range in all cases.

9. The total pulmonary blood flow and the pulmonary arterial pressures measured in 2 cases in the resting state were normal.

Comment.—It is apparent from these results that in this group, studied shortly after the resection of one lung, a slight degree of pulmonary distention was present at the time of study. A combination of reduction in maximum breathing capacity and of hyperventilation reduced the breathing reserve to one-half of normal. Readjustments in the distribution of alveolar ventilation and perfusion through the remaining lung were not perfect; compared with a normal lung a slightly greater proportion of alveoli were well perfused but poorly ventilated and also a slightly greater proportion of alveoli were well ventilated but poorly perfused. Finally, there was no evidence of hypertension in the pulmonary circulation at rest, indicating that the vascular bed of the remaining lung was able to accommodate a twofold increase in pulmonary blood flow in a normal manner.

Individual Cases.—Study of the data in the individual cases of this group shows that the physiologic adjustments, apparently influenced by several factors, were quite variable.

1. In two patients, J. Ra. and C. Ro., aged 15 and 22 years respectively, all the data indicate that the remaining lung was in all respects normal (Fig. 2). In both patients resection of the contralateral lung had been performed for bronchial stenosis of tuberculous origin and had been followed by thoracoplasty.

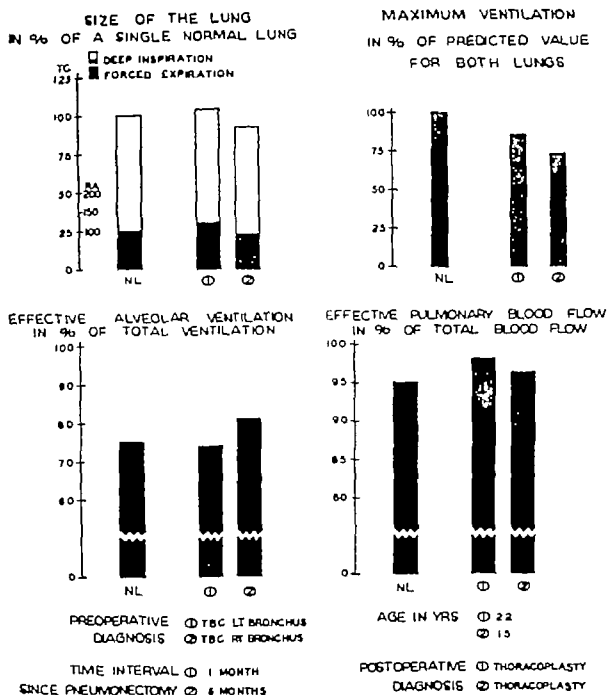


Fig. 2.—Effective alveolar ventilation and pulmonary blood flow in 3 cases with no distention of the lung, studied shortly after pneumonectomy. (1) Case C. Ro. and (2) Case J. Ra.

After pneumonectomy and thoracoplasty the size of the remaining lung during deep inspiration and forced expiration was entirely comparable with that of a homologous normal lung. The maximum breathing capacity was, respectively, 73 and 85 per cent of the predicted value for both lungs in a normal subject. The breathing reserve during exercise and the first minute of recovery was significantly larger than the average value for the group. The arterial blood oxygen saturation at rest and following exercise was well within normal limits.

The effective pulmonary blood flow at rest was in these two patients, respectively, 98 and 96.1 per cent of the total pulmonary blood flow, and during a steady state of moderate exercise increased to 98.5 per cent of the total flow in the case of C. Ro. (Fig. 8). The effective alveolar ventilation was, respectively, 74 and 81 per cent of the total ventilation. All these figures are well within the normal range.

Pulmonary blood flow and pulmonary arterial blood pressures measured in the case of C. Ro. were also normal at rest.

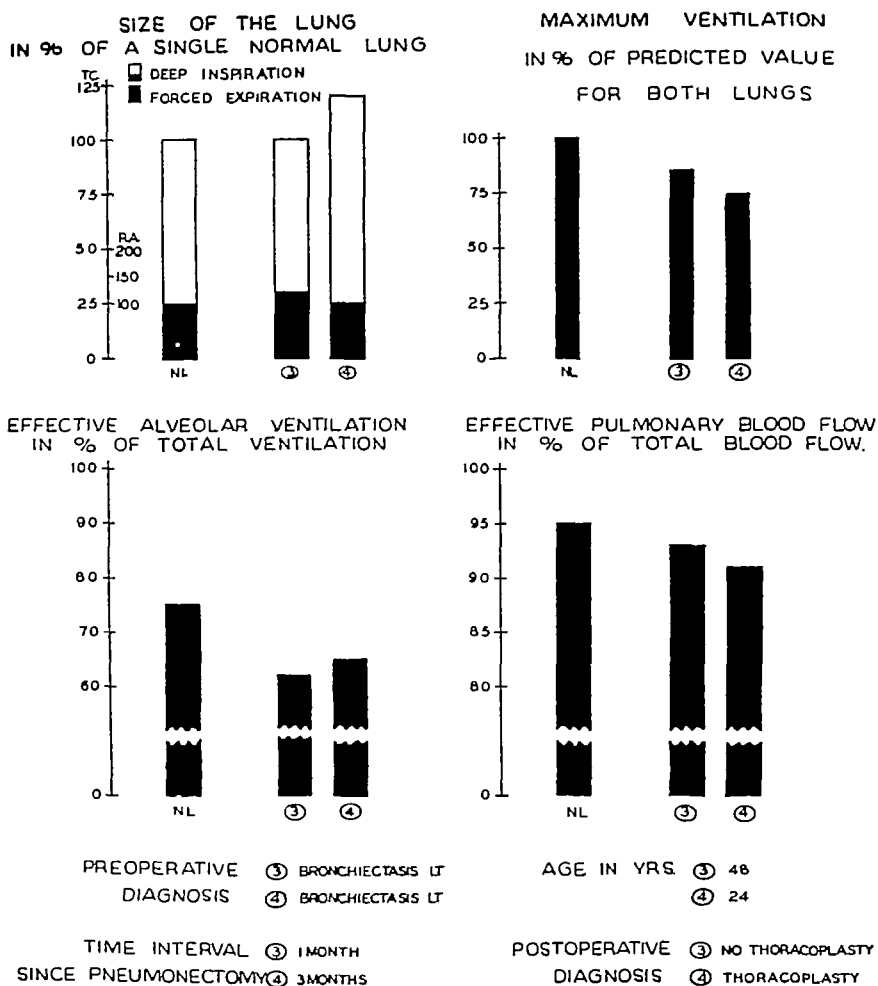


Fig. 3.—Effective alveolar ventilation and pulmonary blood flow in 2 cases with no distention of the lung, studied shortly after pneumonectomy. (3) Case G. H. and (4) Case A. Am.

In summary, the remaining lung in both these cases was not distended and under the conditions of study behaved in an entirely normal manner.

2. In two other patients, A. Am. and G. H., aged 24 and 48 years respectively, the data show that abnormalities in the distribution of alveolar ventilation and perfusion were present (Fig. 3). In these 2 patients the contralateral lung

had been resected for chronic suppuration, associated with organizing pneumonia and fibrosis, and in one case the pneumonectomy was supplemented by thoracoplasty.

Within the next several weeks the remaining lung in both these cases had not become distended, as shown by the measurements of lung volume. The maximum breathing capacity was, respectively, 74 and 84 per cent of the predicted value for two lungs. The breathing reserve was as large during the standard exercise test and the first minute of recovery as in the two previous cases. In one patient the arterial oxygen saturation was slightly below the lower level of normal at rest, and the carbon dioxide tension was normal.

Respiratory gas tension studies, however, indicate that the effective pulmonary blood flow was reduced to, respectively, 92.9 and 91 per cent of total blood flow, and that the effective alveolar ventilation was also reduced to, respectively, 62 and 65 per cent of total ventilation.

The pulmonary blood flow and pulmonary arterial pressure measured in G. H. were normal at rest and during a steady state of moderate exercise, the pulmonary arterial pressure increasing by only 3 mm. Hg.

In summary, there was evidence that the redistribution of alveolar ventilation and perfusion throughout the remaining lung was somewhat impaired. The question is raised, whether in the absence of pulmonary distention these minor changes might not be related to the presence of limited pathologic changes in the remaining lung and were already present before pulmonary resection.

3. In the 3 remaining patients of this group, C. McL., J. Rut., and V. Ab., aged 34, 57, and 35 years, respectively, the data show that the remaining lung was distended or emphysematous and that the alveolar ventilation-perfusion relationships were abnormal (Fig. 4). In these 3 patients the contralateral lung had been resected for a chronic suppurative process associated with organizing pneumonia and fibrosis. In C. McL. and J. Rut. thoracoplasty was performed a short time after pneumonectomy and in both was complicated by a significant degree of scoliosis. In V. Ab. the roentgenograms showed a considerable shift of the mediastinal contents, and the physiologic studies preceded the correction of this shift by thoracoplasty.

Within the next three to five months the remaining lung, as shown by lung volume measurements, was considerably larger than a normal homologous lung in forced expiration, while the size in deep inspiration was lower than normal in one, approximately normal in another, and 150 per cent of normal in the third. Pulmonary distention was therefore present in all 3 cases. The maximum breathing capacity was, respectively, 38, 49, and 31 per cent of the predicted value for both lungs, much lower than in the previous 4 cases. The index of intrapulmonary mixing in V. Ab. was unusually high for a single lung. Hyperventilation was present at rest in the 3 patients and during exercise and recovery in 2 of them; in the third (V. Ab.) relative hypoventilation was observed during these two periods. The arterial oxygen saturation was slightly reduced in all 3 cases, either at rest or following exercise, or in both these states.

Respiratory gas tension studies showed that the calculated effective pulmonary blood flow was, respectively, 92.0, 92.8, and 91.2 per cent of the total blood flow, and that the effective alveolar ventilation was 67, 68, and 67 per cent of the total ventilation.

No observations on the state of the pulmonary circulation are available in any of these cases.

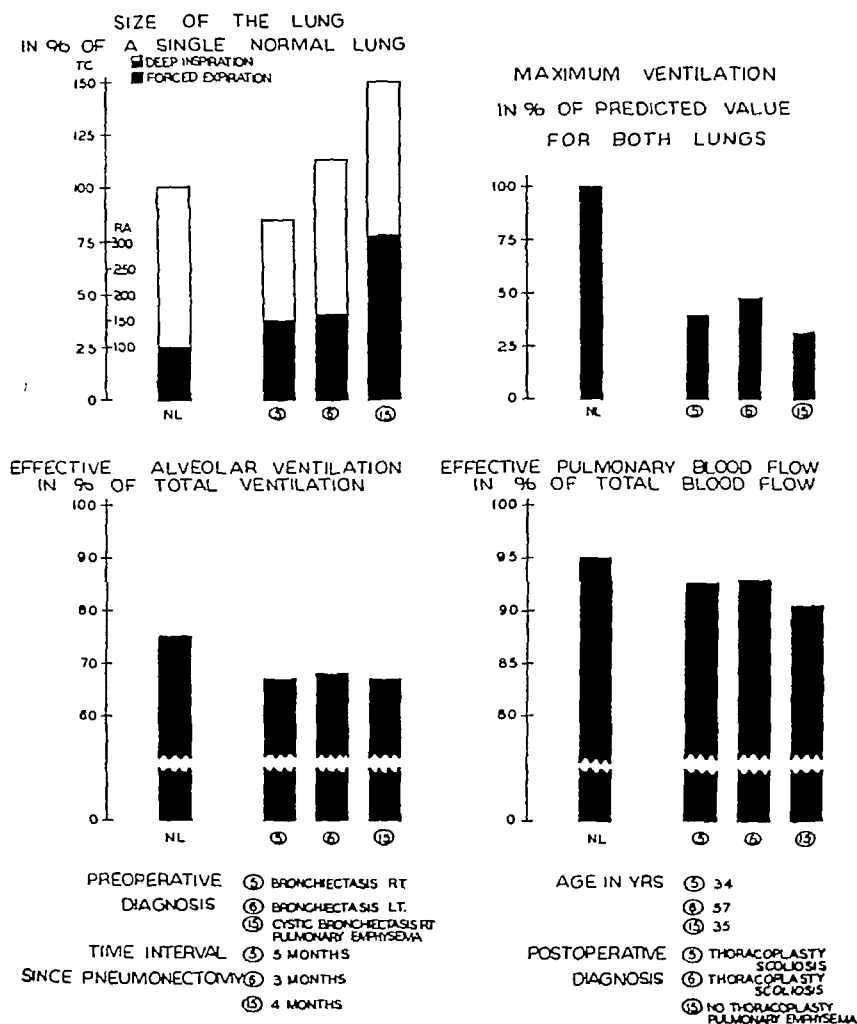


Fig. 4.—Effective alveolar ventilation and pulmonary blood flow in 3 cases with marked distention of the lung, studied shortly after pneumonectomy. (5) Case C McL., (6) J. Rut. and (7) V. Ab.

In summary, the remaining lung in these 3 cases was distended or emphysematous. This condition was accompanied by marked disturbance in the mechanics of the chest bellows, by a considerable reduction in the available breathing reserve, and by some evidence that the distribution of alveolar ventilation and alveolar perfusion was not homogeneous, requiring therefore a large total ventilation to supply adequate alveolar ventilation and causing the arterial blood oxygen saturation to be slightly reduced. The hypothesis that the state of pul-

monary distention might be related to the development of scoliosis, which complicated the thoracoplasties in 2 cases, is supported by the physiologic observations made in one of them before pneumonectomy. The data obtained preoperatively in this case do not suggest the presence of pulmonary distention or emphysema. In contrast, the preoperative studies in the third case, V. Ab., showed that the total capacity was considerably reduced, and that the ratio residual air/total capacity $\times 100$ was very high. Pathologic sections of the resected lung explain in part these findings, as they demonstrated the presence of multiple and large air cysts. The size of the remaining lung postoperatively was markedly increased in both deep inspiration and forced expiration, which finding is explained in part by the marked mediastinal shift observed in roentgenograms and possibly also by the presence of a mild degree of emphysema.

Group II

The data obtained in the 9 patients of this group are tabulated in Tables VIII to XV. Observations were repeated at intervals during several years following resection of one lung. The tables list only the measurements which have not been previously reported. The data on the ventilation-perfusion relationships and on the dynamics of the pulmonary circulation were collected only recently at the time of the latest complete studies listed. In the case of D. M. no such data are available.

Average Findings.—Considered as a group and compared with normal standards the 9 subjects studied late after pneumonectomy showed the following findings.

1. The average size of the remaining lung, in deep inspiration, was 125 per cent of the predicted size of a normal homologous lung in a state of maximum inflation, while its average size in forced expiration was 175 per cent of that of a normal lung in the state of maximum deflation and the ratio residual air/total capacity $\times 100$ was somewhat higher than normal.

2. The maximum breathing capacity was reduced to only 70 per cent of the predicted value for a normal chest with both lungs.

3. A small degree of hyperventilation was present during the three periods of observation at rest, during the standard exercise test, and during the first minute of the recovery period. The breathing reserve during the standard exercise test and the first minute of the recovery period was, respectively, 53 and 47 liters, or approximately 60 per cent of the predicted values.

4. The mean index of intrapulmonary mixing was normal.

5. The mean oxygen consumption at rest and during the standard one-minute exercise test was normal.

6. The mean arterial oxygen saturation was approximately normal at rest and following exercise was slightly below the lower range of normal.

7. The CO_2 tension in the arterial blood, while breathing room air, was normal at rest and during a steady state of bicycle exercise. The oxygen tension was slightly lower than normal in both these states, and the alveolo-arterial pO_2 gradient was at the upper limit of normal. The effective pulmonary blood flow and the effective alveolar ventilation were, respectively, 93.7

TABLE VIII. LUNG VOLUMES AND MAXIMUM BREATHING CAPACITY IN 9 CASES STUDIED SEVERAL YEARS AFTER PNEUMONECTOMY

SUBJECT	TIME AFTER OPERATION (MO.)	VITAL CAPACITY (IN C.C.)		RESIDUAL AIR (IN C.C.)		TOTAL CAPACITY (IN C.C.)		RESIDUAL AIR TOTAL CAPACITY $\times 100$		MAXIMUM BREATHING CAPACITY (IN LITERS)	
		PRE-DICTED*	OB-SERVED	PRE-DICTED*	OB-SERVED	PRE-DICTED*	OB-SERVED	PRE-DICTED	OB-SERVED	PRE-DICTED†	OB-SERVED
H. W.	131	1820	2910	455	628	2275	3338	20	18	102	86
R. G.	108	2447	3623	611	700	3060	4323	20	16	135	102
S. R.	25	1800	2901	550	819	2350	3720	23	22	124	92
J. Lo.	104	2480	2537	620	1054	3100	3591	20	29	136	76
J. Rus.	1	1485	1912	663	1180	2148	3092	31	38	80	69
	26	1485	1616	663	1374	2148	2990	31	46	80	74
V. Wat.	17	1854	2201	825	976	2679	3147	31	31	89	76
	42	1854	2229	825	1004	2679	3294	31	31	89	65
	50	1854	2154	825	1682	2679	3836	31	44	89	65
	91	1854	2017	825	1590	2679	3607	31	44	79	75
D. M.	13	1990	1930	890	1478	2880	3474	31	43	96	41
	50	1990	1566	890	2247	2880	3985	31	56	96	24
E. Re.	80	2254	2589	687	1139	2941	3728	23	31	104	68
F. I.	15	1733	2396	772	865	2505	3261	31	27	114	73
	77	1733	2342	772	1213	2505	3555	31	34	114	79
Average of 9 cases after pneumonectomy†	79	1960	2450	685	1103	2644	3630	26	32	108	75

*Predicted volume of a homologous single normal lung.

†Predicted maximum breathing capacity for a normal subject with intact chest and lungs and of the same age, sex, and size.

‡Includes only the latest series of measurements in each case when several studies were made.

TABLE IX. (A) INDEX OF INTRA-PULMONARY MIXING; (B) VENTILATION AND BREATHING RESERVE AT REST, DURING A ONE-MINUTE STANDARD EXERCISE AND DURING THE FIRST MINUTE OF RECOVERY IN 9 CASES STUDIED SEVERAL YEARS AFTER PNEUMONECTOMY

SUBJECT	TIME AFTER OPERATION (MO.)	INDEX OF INTRA-PULMONARY MIXING (N.%)	BASAL				VENTILATION (IN LITERS PER MIN. PER M ² B.A.)						BREATHING RESERVE (IN LITERS PER MIN.)					
			PRE-DICTED		OBSERVED		EXERCISE		RECOVERY			EXERCISE		RECOVERY				
			PRE-DICTED	OBSERVED	PRE-DICTED	OBSERVED	PRE-DICTED	OBSERVED	PRE-DICTED	OBSERVED		PRE-DICTED	OBSERVED	PRE-DICTED	OBSERVED			
H. W.	131	.89	3.2	3.7	9.0	10.1	10.9	11.4	87	60		87	60	79	65			
R. G.	108	1.21	3.0	4.0	11.0	14.0	12.5	12.9	115	61		115	61	106	81			
S. Ro.	25	.91	3.1	4.5	10.0	14.0	13.4	12.1	105	65		105	65	92	52			
J. Lo.	104	1.37	3.0	3.9	11.0	12.5	11.9	11.9	116	54		116	54	104	51			
J. Ru.	1	.63	3.9	4.2	11.2	16.3	14.5	20.6	64	45		64	45	55	39			
	26	.84	3.9	6.2	11.2	22.5	14.5	25.6	64	42		64	42	55	29			
V. Wat.	17	1.02	3.9	4.3	11.2	11.0	14.5	14.4	60	44		60	44	59	30			
	42	1.43	-	-	-	-	-	-	-	-		-	-	-	-			
	50	1.18	3.9	4.4	11.2	16.5	14.5	17.7	68	53		68	53	58	32			
	91	1.28	3.9	6.3	11.2	17.5	14.5	20.7	61	47		61	47	52	31			
D. M.	13	1.19	3.9	4.1	11.2	17.4	14.5	16.8	76	30		76	30	65	20			
	50	2.98	3.9	5.5	11.2	11.0	14.5	13.0	72	3		72	3	63	4			
E. Ro.	80	-	3.1	3.6	10.0	10.3	13.4	10.3	88	75		88	75	77	69			
F. L.	15	-	3.1	2.5	10.0	12.1	13.4	9.8	90	51		90	51	84	49			
	70	-	3.9	3.2	11.2	9.6	14.5	11.9	88	61		88	61	77	41			
Average of 9 Cases*	77	1.35	3.6	4.5	10.6	13.6	13.4	14.4	88	53		88	53	78	47			

*Includes only the latest series of measurements in each case, when several studies were made.

TABLE X. OXYGEN CONSUMPTION AND ARTERIAL BLOOD OXYGEN SATURATION AT REST, DURING AND FOLLOWING A ONE-MINUTE STANDARD EXERCISE IN 9 CASES STUDIED SEVERAL YEARS AFTER PNEUMONECTOMY

SUBJECT	TIME AFTER OPERATION (MO.)	OXYGEN CONSUMPTION				ARTERIAL BLOOD OXYGEN SATURATION (IN %)	
		IN C.C. PER MIN. PER M ² B.S.		IN C.O PER LITER OF VENTILATION		BASAL	RECOVERY
		REST	EXERCISE	REST	EXERCISE		
Normal		132	495	44.5	55.4	96.0	96.0
H. W.	131	127	476	41.8	56.8	95.0	96.5
R. G.	108	143	519	37.4	45.7	96.2	95.0
S. Ro.	25	146	564	38.8	48.8	95.5	—
J. Lo.	104	132	502	44.6	63.2	97.0	96.0
J. Rus.	1	131	552	36.7	39.4	97.2	97.5
	26	146	586	28.0	31.0	98.0	96.2
V. Wat.	17	116	416	31.0	42.8	93.5	93.6
	42	—	—	—	—	—	—
	50	111	429	29.1	30.4	95.0	97.0
	91	141	427	26.9	29.5	96.0	96.0
D. M.	13	134	512	34.2	34.0	95.6	90.8
	50	126	345	27.0	35.1	91.6	85.4
E. Re.	80	119	548	40.4	64.8	94.4	91.8
F. I.	15	102	442	48.2	42.6	96.6	97.9
	79	119	342	45.2	42.8	95.2	91.0
Average of 9 cases*	77	133	479	36.7	44.2	95.4	93.1

*Includes only the latest series of measurements in each case, when several studies were made.

and 70 per cent of the total blood flow and of the total ventilation in the resting state. In 3 of the cases the measurements repeated during a steady state of moderate exercise showed a significant increase: the effective pulmonary blood flow rose to 97.9 per cent of the total blood flow while the effective alveolar ventilation constituted 79 per cent of the total ventilation.

8. The mean alveolo-arterial pO_2 gradient of the group while breathing a low oxygen mixture was within the normal range.

9. The average pulmonary blood flow and mean pulmonary arterial pressure were within the normal range at rest. During the steady state of moderate exercise the pulmonary arterial pressure rose from 15 mm. Hg at rest to the abnormally high value of 24 mm. Hg.

Comment.—From these results it appears that in this group studied at an average time of seventy-seven months after the resection of one lung, the remaining lung was larger than normal in deep inspiration and was distended in forced expiration. Discussion as to whether the latter finding indicates that a mild degree of chronic pulmonary emphysema was present will be postponed until after presentation of the individual cases.

Compared with the average maximum breathing capacity in the group studied soon after pneumonectomy, the average maximum breathing capacity was reduced less, in spite of a state of greater pulmonary distention, and the breathing reserve was larger during standard exercise and the first minute of recovery. Minor alterations in the distribution of alveolar ventilation and alveolar perfusion were also present. The dynamics of the pulmonary circulation were normal at rest, as in Group I. However, in the course of a moderate exercise the pulmonary arterial pressure became abnormally high. In this state the

TABLE XI. CO₂ AND O₂ PRESSURE GRADIENTS BETWEEN AMBIENT AIR, ALVEOLAR AIR, AND ARTERIAL BLOOD IN 8 CASES STUDIED SEVERAL YEARS AFTER PNEUMONECTOMY

SUBJECT	TIME AFTER OPERATION (MO.)	STATE	O ₂ TENSIONS (PO ₂) (IN MM. HG.)				PO ₂ GRADIENTS (IN MM. HG.)		CO ₂ TENSIONS (PCO ₂) (IN MM. HG.)		TIDAL AIR (IN C.C.)	RESPIRATION QUOTIENT
			INSPIRED AIR (I)	ALVEOLAR AIR (AL)	ARTERIAL BLOOD (AR)	I-AL	I-AR	AT-AR	EXP. AIR	ART. BLOOD		
H. W.	131	Rest (R. A.)	151	100	91	51	9	9	21	36	369	.70
	131	Rest (low O ₂)	89	55	43	34	12	12	20	32	428	.95
	134	Rest* (R. A.)	153	106	94	47	12	12	19	33	540	.71
	134	Exer.* (L)	152	117	96	35	21	21	27	33	780	.94
R. G.	108	Rest (R. A.)	148	90	92	50	4	4	22	42	364	.84
	108	Exer. (B)	148	100	90	48	10	10	31	43	1120	.90
	110	Rest (low O ₂)	82	48	42	34	6	6	22	38	303	1.11
	110	Rest* (R. A.)	158	107	104	51	3	3	22	41	332	.81
S. Ro.	110	Exer.* (L)	157	110	110	47	6	6	29	37	645	.93
	25	Rest (R. A.)	151	109	85	42	14	14	23	35	506	.83
	25	Exer. (B)	150	112	90	38	16	16	28	35	1170	.91
	25	Rest (low O ₂)	85	49	41	36	8	8	21	32	570	.93
J. Lo.	104	Rest (R. A.)	150	100	79	50	21	21	29	40	478	.92
	104	Exer. (B)	151	103	78	48	25	25	34	40	745	.84
	104	Rest (low O ₂)	89	48	41	41	7	7	27	42	412	1.02
	26	Rest* (R. A.)	153	87	82	66	5	5	15	46	480	.70
J. Rus.	26	Rest (R. A.)	153	93	88	60	3	3	16	49	510	.81
	26	Rest (low O ₂)	90	54	39	30	15	15	16	44	598	1.21
	91	Rest (R. A.)	150	97	86	53	11	11	15	40	390	.75
	91	Exer. (B)	149	111	92	58	19	19	16	36	929	.95
V. Wat.	91	Rest (low O ₂)	101	50	45	45	11	11	14	35	427	.77
	92	Rest* (R. A.)	150	90	89	51	10	10	15	41	374	.81
	92	Exer.* (L)	149	104	90	45	14	14	22	45	653	1.00
	81	Rest (R. A.)	152	95	79	57	16	16	18	40	398	.81
E. Ro.	81	Exer. (B)	153	100	81	47	25	25	34	39	1040	.83
	81	Rest (low O ₂)	80	44	37	42	7	7	20	43	500	.91
	79	Rest (R. A.)	151	101	91	50	10	10	26	40	548	.80
	79	Exer. (B)	151	101	91	50	10	10	32	42	1230	.84
F. I.	79	Rest (low O ₂)	85	54	37	31	17	17	26	32	792	1.03

*Respiratory gas tensions studied simultaneously with blood flow measurements by cardiac catheterization technique.

Rest (R. A.), resting state, breathing room air.

Rest (low O₂), resting state, breathing a mixture of 12 per cent oxygen in nitrogen.

Exer. (B), bicycle exercise with steady state.

Exer. (L), steady state of a leg exercise with subject flat on back.

For averages see Table XII.

TABLE XII. AVERAGE OXYGEN TENSION GRADIENTS BETWEEN INSPIRED AIR, ALVEOLAR AIR, AND ARTERIAL BLOOD AND AVERAGE ARTERIAL CARBON DIOXIDE TENSIONS IN TWO GROUPS OF SUBJECTS AFTER PNEUMONECTOMY

STATE	GROUP	NO. OF CASES	O ₂ TENSIONS (pO ₂) (IN MM. HG)			pO ₂ GRADIENTS (IN MM. HG)		CO ₂ TENSIONS (pCO ₂) (IN MM. HG)
			INSPIRED AIR (I)	ALVEOLAR AIR (AL)	ARTERIAL BLOOD (AR)	I-AL	AL-AR	ARTERIAL BLOOD
Rest, R. A. breathing	Normal	8	152	104	94	48	10	39
	Group I	7	151	102	87	49	15	39
	Group II	8	152	100	89	52	12	41
Rest, low O ₂ breathing	Normal*	3	84*	42*	33*	42*	9*	38*
	Group I	6	85	50	41	35	9	34
	Group II	8	88	51	41	37	10	37
Exercise, bicycle, steady state	Normal	8	151	109	95	42	14	38
	Group I	3	150	110	94	40	16	38
	Group II	6	150	106	88	44	18	39

Arterial blood CO₂ and oxygen tension measurements with low oxygen breathing made in only 3 of the 8 normal physicians recently studied. In a much larger control group previously studied by Lillenthal and associates similar values were obtained.

TABLE XIII. EFFECTIVE ALVEOLAR VENTILATION AND EFFECTIVE PULMONARY BLOOD FLOW IN 7 CASES STUDIED SEVERAL YEARS AFTER PNEUMONECTOMY

SUBJECT	STATE	ALV. ARTERIAL pO ₂ GRADIENT (MM. HG)	PULMONARY BLOOD FLOW		RATIO B A	PULMONARY VENTILATION		RATIO D C
			TOTAL (A) (LITERS PER MIN. PER M ² B.S.)	EFFECTIVE (B) (LITERS PER MIN. PER M ² B.S.)		TOTAL (C) (LITERS PER MIN. PER M ² B.S.)	EFFECTIVE ALV. (D) (LITERS PER MIN. PER M ² B.S.)	
Normal	Rest	<12	3.10	2.95	>95.0	3.82	2.97	>70
H. W.	Rest	12	3.44	3.29	95.4	4.26	2.94	69
	Exercise	21	5.15	5.00	97.1	9.48	8.75	91
R. G.	Rest	3	3.17	3.11	98.0	4.04	2.99	74
	Exercise	6	6.24	6.19	99.1	12.40	10.90	88
S. Ro.	Rest	14	-	-	94.7*	4.53	3.81	79
J. Lo.	Rest	21	2.94	2.67	90.6	3.86	3.01	78
J. Ru.	Rest	5	3.15	3.05	95.0	6.08	3.70	61
V. Wat.	Rest	10	2.29	2.21	96.7	5.63	2.98	53
	Exercise	14	3.24	3.13	97.6	10.76	6.25	58
E. Re.	Rest	16	3.52	3.05	86.8	3.41	2.52	74
F. I.	Rest	10	2.94	2.74	93.2	3.15	2.42	77
Average of 8 cases at rest		11	3.06	2.87	93.7	4.36	3.05	70
Average of 3 cases during exercise		14	4.88	4.77	97.9	10.88	8.63	79

*Mixed venous blood not collected. Mixed venous blood O₂ saturation per cent assumed to be 25 per cent lower than arterial blood saturation.

TABLE XIV. EFFECTIVE ALVEOLAR VENTILATION AT REST AND DURING THE STEADY STATE OF A BICYCLE EXERCISE IN A GROUP OF 8 CASES AFTER PNEUMONECTOMY

STATE	GROUP	NO. OF CASES	ALV. VENTILATION (LITERS PER MIN. PER M ² B.S.)	ALV. VENTILA- TION (LITERS PER MIN. PER M ² B.S.)	O ₂ INTAKE (C.C. PER MIN. PER M ² B.S.)	O ₂ INTAKE (C.C. PER LIT.) ALV. VENTILA- TION
			$\frac{\text{ALV. VENTILATION}}{\text{TOTAL VENTILATION}} \times 100$			
Rest	Normal	8	78	2.97	141	48
	Pneumectomy Groups I*-II	8	71	3.16	145	46
Exercise, steady state	Normal	8	88	12.80	567	44
	Pneumectomy Groups I*-II	8	75	10.95	523	48

*This mixed group includes the cases A. Am. and C. McL. from Group I and all the cases from Group II except H. W.

pulmonary blood flow through the remaining lung was larger than the blood flow through each lung of a normal individual performing a very severe type of exercise. Under such circumstances the pulmonary arterial pressure of the normal individual remains normal.^{7,8} The various mechanisms involved in the abnormal increase of the pulmonary arterial pressure will be discussed later.

Individual Cases.—Examination of the data in the individual cases of this group reveals marked variations in their physiologic status.

1. In 3 patients, S. R., H. W., and R. G., aged 41, 17, and 21 years, respectively, the data indicate that the remaining lung was normal as far as can be judged from the studies (Fig. 5). Two of the cases, H. W., and R. G., have already been discussed in two previous reports.^{1,2} The data concerning the alveolar ventilation-perfusion relationships and the dynamics of the circulation obtained recently confirm and supplement the general impression reached on the basis of the previously reported less complete studies of pulmonary function. In these 3 patients the resection had been performed for chronic suppuration of one lung and had not been followed by thoracoplasty.

It is quite remarkable that the size of the lung in these 3 subjects at the end of the deepest inspiration was approximately 150 per cent of that of a normal homologous lung in the same respiratory position. This finding was obviously due to a mediastinal shift which is well known to occur during deep inspiration. However, in forced expiration the size of the deflated lung was about that of a normal homologous lung, as shown by the ratio residual air/total capacity $\times 100$, which was, respectively, 22, 18, and 16. The maximum breathing capacity was, respectively, 74, 84, and 76 per cent of the predicted value for both lungs and the breathing reserve in the absence of any significant degree of hyperventilation during the standard exercise test and the first minute of recovery was very large, ranging from 60 to 80 per cent of predicted normal values. The arterial oxygen saturation at rest and during exercise was within the normal range of variation in all 3 cases.

Studies of the ventilation-perfusion relationships in these 3 subjects demonstrated that the effective pulmonary blood flow was, respectively, 94.7, 95.4, and 97 per cent of the total pulmonary blood flow and that the effective alveolar

TABLE XV. PULMONARY BLOOD FLOW AND PULMONARY ARTERIAL PRESSURES AT REST AND DURING THE STEADY STATE OF A MODERATE EXERCISE*
IN 9 CASES AFTER PNEUMONECTOMY

SUBJECT	TIME AFTER OPERA- TION (MO.)	STATE	PULSE RATE	O ₂ CON- SUMP- TION (C.C. PER MIN. PER M ² B.S.)	ARTERIAL VENOUS O ₂ DIP- PERENCE (C.C. PER LITERS)	CARDIAC OUTPUT		ARTERIAL BLOOD PRESSURES				SYSTEMIC VASCULAR RESIST- ANCE DYNES CM. ⁻⁵ SEC. M ² B.S.
						LITERS PER MIN. PER M ²	S.V.	PULMONARY		SYSTEMIC		
								S/D (MM. HG)	MEAN (MM. HG)	S/D (MM. HG)	MEAN (MM. HG)	
NNormal (average of 3)		Rest†		126	39	3.27	83	20/9	13	112/65	84	2090
	I	Exer. (B)†	406	73	5.40	104	23/4	11	125/78	100	1515	
		Rest	145	31	4.68	74	25/6	14	130/72	93	1980	
C. Ro.	1½	Exer. (L)	132	268	47	5.57	76	35/8	17	160/86	109	1960
		Rest	96	135	42	3.21	47	24/9	14	127/85	102	2550
		Exer. (B)	133	315	72	4.37	52	-	-	145/95	114	2090
H. W.	134	Rest	73	127	37	3.44	89	18/9	12	114/65	81	2500
	Exer. (L)	108	319	62	5.15	85	23/14	17	142/82	98	2000	
R. G.	110	Rest	65	127	40	3.17	81	23/7	12	116/71	89	2240
	Exer. (L)	105	443	71	6.24	105	35/19	24	152/84	110	1410	
J. Lo.	104	Rest	70	165	56	2.94	77	27/-	-	130/81	94	2560
	26	Rest	101	144	46	3.15	45	36/8	19	115/68	89	2260
J. Ru.	92	Rest	98	119	52	2.29	39	30/13	19	151/91	111	3880
		Exer. (L)	108	259	80	3.24	50	44/24	31	195/113	140	3460
E. Re.	81	Rest	61	123	35	3.52	95	24/7	12	117/62	89	2020
	Exer. (L)	102	277	55	5.04	80	34/11	18	125/68	95	1510	
F. I.	79	Rest	59	112	38	2.94	94	29/11	16	143/69	101	2750
		Exer. (L)	97	196	56	3.50	68	55/17	28	171/96	128	2930
Average of 9 cases at rest			82	133	42	3.27	71	26/9	15	127/74	95	2526
Average of 7 cases at rest			81	127	39	3.32	74	25/9	14	128/74	95	2560
Average of 7 cases during exercise			112	297	63	4.74	74	38/16	23	155/89	113	2193

*Measurements started after 10 minutes of a moderate leg exercise with subject flat on back, except in Case C. Ro. where the measurements were started during the tenth minute of a moderate bicycle exercise.

†Data obtained on 3 normal physicians.³

ventilation was, respectively, 79, 74, and 69 per cent of the total ventilation. All these figures are normal. Observations made during a steady state of moderate exercise in 2 of the subjects (Fig. 8) showed a marked increase in the efficiency of alveolar perfusion and ventilation. The pulmonary membrane diffusion gradient was normal.

The pulmonary blood flow and the pulmonary arterial blood pressure measured in H. W. and R. G. were normal at rest. In the course of moderate exercise during which the pulmonary blood flow was nearly twice the resting blood flow and significantly larger than the volume of blood flowing through each lung of a normal individual during a severe type of exercise, the mean pressure rose from 12 mm. Hg at rest to 17 and 24 mm. Hg, respectively.

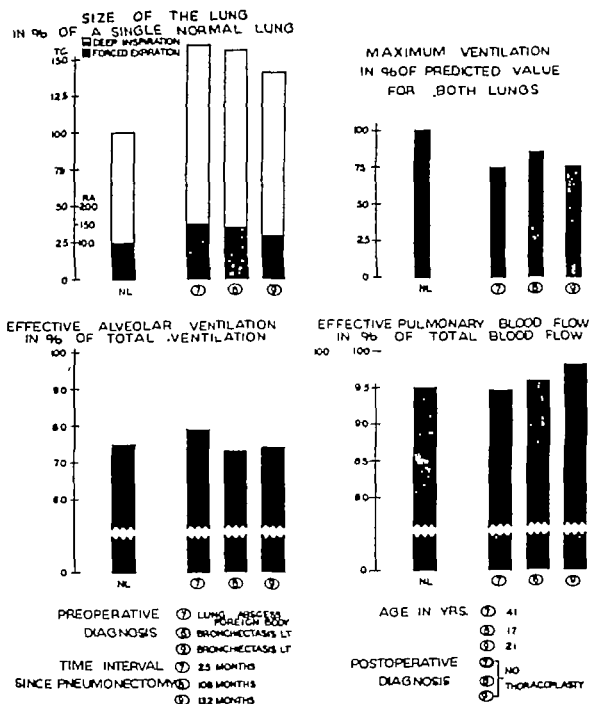


Fig. 5.—Effective pulmonary blood flow and alveolar ventilation in 3 cases with no lung distention, studied several years after pneumonectomy. (7) Case S. Ro., (8) Case H. W., and (9) Case R. G.

In summary, the remaining lung in these 3 cases, although larger than normal in the state of maximum inflation, functioned normally. The physical activity of these 3 subjects was not limited, the reduction in breathing capacity

was slight, and a study of the relationships between alveolar ventilation and alveolar perfusion revealed these to be entirely normal at rest as well as during moderate exercise. This latter finding is of interest since during this exercise the total ventilation and total pulmonary blood flow through the remaining lung reached greater values than those hitherto observed through each lung of normal individuals during a severe exercise test. The only abnormal finding was the observation of a mild degree of pulmonary hypertension during the performance of the exercise. The meaning of this observation will be considered later in the discussion.

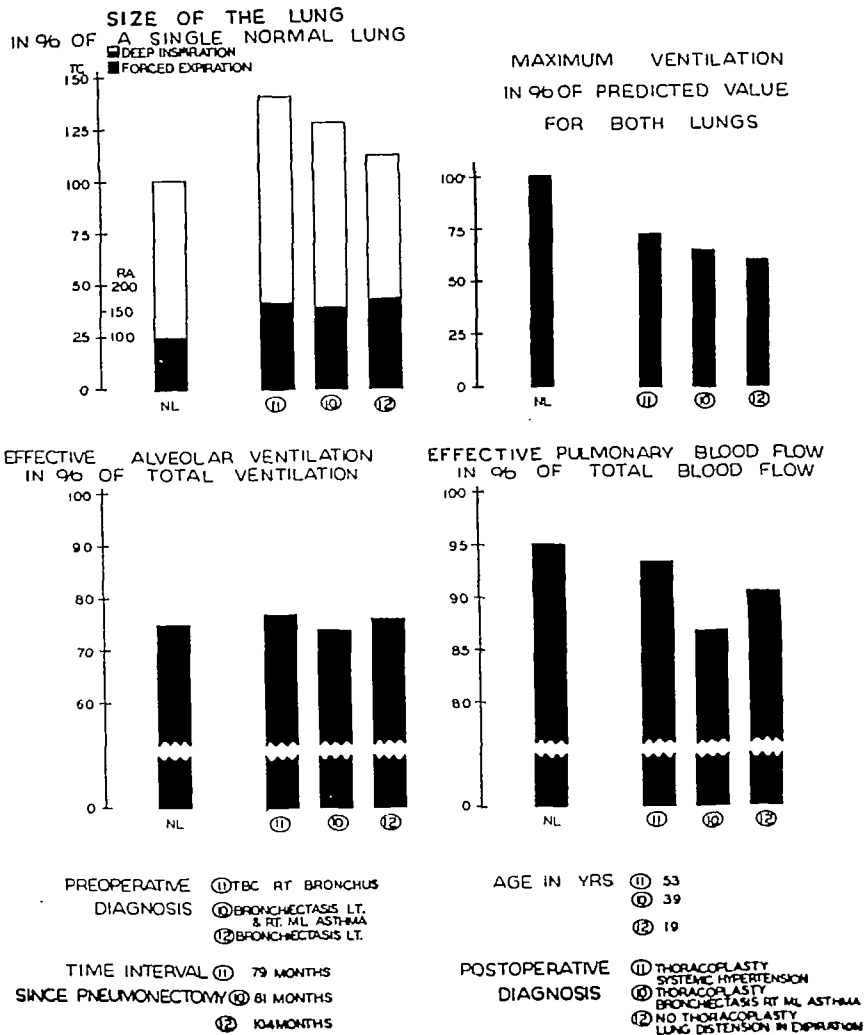


Fig. 6.—Effective pulmonary blood flow and alveolar ventilation in 3 cases with moderate lung distention, studied several years after pneumonectomy. (10) Case E. Re., (11) Case F. I., and (12) Case J. Lo.

2. In 3 patients, F. I., E. Re., and J. Lo., aged 53, 39, and 19 years, respectively, the data show a small degree of pulmonary distention associated with slight abnormalities in the distribution of alveolar ventilation and alveolar perfusion in the remaining lung (Fig. 6). All of these cases have already been dis-

cussed in 3 previous reports^{1, 2, 3} on the basis of less complete data. The resection of the lungs in E. Re. and J. Lo. had been performed for chronic suppuration of one lung; in addition E. Re. showed, and still shows, evidence of bronchiectasis in the middle lobe of the remaining right lung, and is suffering from persistent but mild asthma. In F. I. the resection of the right lung was done for stenosis of the right main bronchus of tuberculous origin, which caused over a period of twenty years acute and repeated episodes of bronchial obstruction. In E. Re. and F. I. the pneumonectomy was followed by thoracoplasty.

In these 3 subjects the size of the remaining lung compared with that of a normal homologous lung in the same state was increased in deep inspiration, more so in F. I., less so in J. Lo. In contrast to the 3 previous cases, however, the size of the lung in forced expiration is significantly greater than that of a normal lung in the same state, the state of moderate distention being the greatest in J. Lo., the least in F. I. The maximum breathing capacity was in 2 of the 3 cases reduced more than in the 3 previous cases. Its values in F. I., E. Re., and J. Lo., respectively, 73, 65, and 56 per cent of the predicted normal value with both lungs, bearing a striking relationship to the degree of distention. Hyperventilation did not develop in any of these cases during or following exercise and the moderate reduction of breathing reserve during the standard exercise test and the first minute of recovery was related to the reduction in maximum breathing capacity. Measurements of the arterial oxygen saturation in F. I. and E. Re. at rest and following exercise were somewhat lower than normal, especially after exercise, suggesting that some poorly ventilated alveoli were still perfused.

More precise methods of studies of the ventilation-perfusion relationships demonstrated that at rest the effective pulmonary blood flow was in F. I., E. Re., and J. Lo., respectively, 93.2, 86.8, and 90.6 per cent of the total pulmonary blood flow. In contrast to these figures, which indicate perfusion of poorly or nonventilated alveoli in varying degree, the effective alveolar ventilation was, respectively, 77, 76, and 78 per cent of total ventilation. These figures indicate that there was no ventilation of poorly perfused areas. The pulmonary membrane diffusion gradient was normal in two of the subjects and slightly elevated in F. I.

The pulmonary blood flow and pulmonary blood pressure were normal at rest in the 3 cases. During moderate exercise the pulmonary arterial pressure rose in E. Re. from 12 to 18 mm. Hg when the pulmonary blood flow was 144 per cent of the resting blood flow, and in F. I. it rose from 16 to 28 mm. Hg when the pulmonary blood flow was only 119 per cent of the resting flow. It should be noted (Table XV) that in the case of F. I. the systemic arterial pressure rose considerably during a steady state of very moderate exercise and that the calculation of the systemic vascular resistance indicated some degree of vasoconstriction rather than the usual vasodilatation observed during exercise. The question is raised, therefore, whether a latent state of systemic hypertension was not revealed during the increased physical activity. The development of pulmonary hypertension, if this assumption is correct, would be the result of an increase in pressure in the pulmonary venous bed due to poorer emptying of the

left ventricle in the face of an increasing peripheral resistance rather than the result of an increased resistance through a pulmonary vascular bed restricted in its distensibility.

In summary, the remaining lung in these 3 cases was not normal. A combination of pulmonary distention and of pathologic changes certainly related in one case, probably related in the 2 other cases, to the chronic process for which the contralateral lung was resected, resulted in the persistence of pulmonary flow through areas of the lung which were not ventilated. In the greater part of the lung, however, the relationship between alveolar ventilation and perfusion was normal. It is of importance to note that observations over a long period of years have not shown any significant change in the degree of pulmonary distention in these 3 cases. This state of distention probably was present shortly after pneumonectomy and might be due partly to an underlying pathologic process. It may have been somewhat accentuated following the resection, but at all events was limited by the fixation of the mediastinum in its normal position with or without the help of thoracoplasty.

3. In the 3 remaining cases, D. M., J. Ru., and V. Wat., aged, respectively, 66, 61, and 72 years, the data show that the remaining lung was the site of chronic pulmonary emphysema of mild to moderate degree. In these 3 cases the lung had been resected for carcinoma of a main bronchus and pneumonectomy was not supplemented by thoracoplasty. The survival times up to the last observations were, respectively, fifty, twenty-six, and ninety-one months. One of these 3 patients (D. M.) died after a seventy-two month survival, following an acute respiratory infection.

Physiologic studies of patient D. M. have been reported previously.³ The reoperative studies showed a marked degree of chronic bilateral pulmonary emphysema of the obstructive type. Postoperative studies fifty months after pneumonectomy indicated that the state of chronic obstructive emphysema had become more severe. The ratio residual air/total capacity $\times 100$ rose from 43 to 56, the maximum breathing capacity decreased from 41 to 24 liters. The breathing reserve during the standard exercise test and the first minute of recovery was negligible and consequently dyspnea was severe and prolonged. The arterial oxygen saturation was reduced to 91.6 per cent at rest and decreased to 85.4 per cent following the standard exercise test. This finding suggests considerable perfusion of poorly ventilated or nonventilated alveoli. The last physiologic studies, performed three years ago, do not include observations on gas tensions performed simultaneously with cardiac catheterization. Hence a quantitative study of the abnormalities in the distribution of alveolar ventilation and perfusion is not available.

In the other 2 cases, J. Ru. and V. Wat., repeated measurements of lung volume have demonstrated the slow development of a moderate type of non-obstructive emphysema which was not present shortly, or even several months, after pneumonectomy (Fig. 7). The size of the remaining lung on deep inspiration was at the time of the last studies 139 and 135 per cent, respectively, of the predicted size of a normal homologous lung in the same state. In forced expiration the size of the lung was 208 and 193 per cent, respectively, of the

predicted size of a normal homologous lung in the same state. Clinical studies and examination of the spiograms⁸ did not reveal any evidence of bronchiolar obstruction. A remarkable finding was that the maximum breathing capacity in these 2 patients was, respectively, 93 and 95 per cent of the predicted values with both lungs. In absolute terms, the maximum breathing capacity was as large as in the 3 previous subjects who were younger and showed a far lesser degree of pulmonary distention in expiration. Marked hyperventilation was

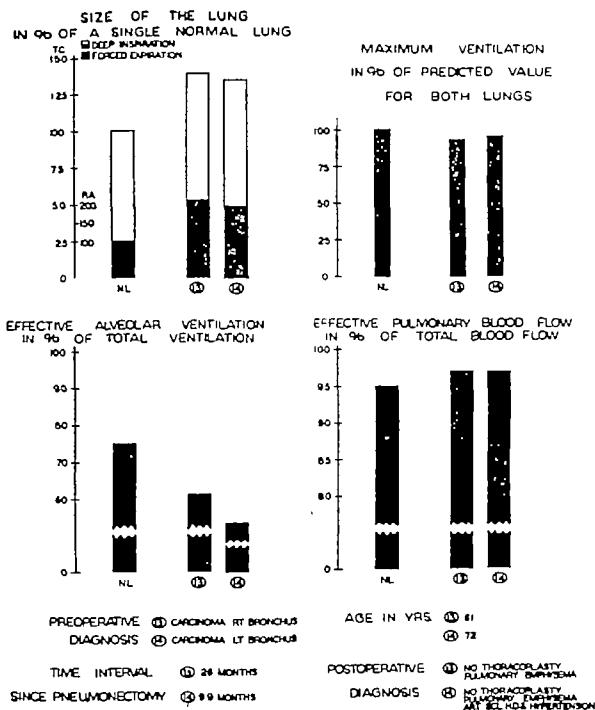


Fig. 7.—Effective pulmonary blood flow and alveolar ventilation in 2 cases of elderly subjects with mild nonobstructive emphysema, studied several years after pneumonectomy. (13) Case J. Rus. and (14) Case V. Wat.

present during the 3 periods of observation. Consequently, the breathing reserve was reduced below normal values. It was approximately 55 per cent of normal during the first minute of recovery following the standard exercise test and mild dyspnea of short duration was present at that time. The index of intrapulmonary mixing was normal. The arterial blood saturation at rest and following the standard exercise test was well within the normal range of variation.

The respiratory gas tension studies demonstrated that at rest the effective pulmonary blood flow was, respectively, 96.7 and 97 per cent of the total pulmonary blood flow. During a steady state of moderate exercise the effective blood flow rose in one patient, V. Wat., to 97.6 per cent of the total blood flow (Fig. 8). These figures indicate that virtually all perfused areas were well ventilated. These gas tension studies revealed, however, that a large proportion of well ventilated alveoli were poorly perfused or nonperfused. The effective alveolar ventilation in per cent of total ventilation reached the lowest figures in the entire group of patients after pneumonectomy, respectively, 61 and 53 per cent. In V. Wat. during the steady state of moderate exercise the effective

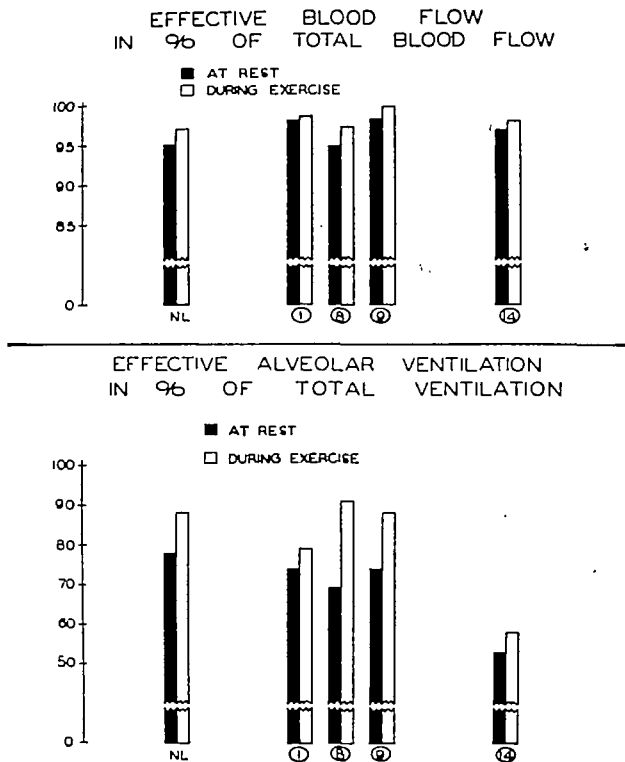


Fig. 8.—Influence of exercise upon effective pulmonary blood flow and alveolar ventilation in 3 young adults with no distention of the lung and in an elderly subject with nonobstructive pulmonary emphysema. (1) Case C. Ro., (8) Case H. W., (9) Case R. G., and (14) Case V. Wat.

alveolar ventilation was still only 58 per cent of the total ventilation (Fig. 9). On the basis of these figures it would appear that the pulmonary vascular bed was restricted in parts of the lung which were still effectively ventilated, although whenever alveolar perfusion persisted the alveoli were well ventilated. The pulmonary membrane diffusion gradient was normal in V. Wat. and very slightly elevated in J. Ru.

Studies of the dynamics of the pulmonary circulation showed definite abnormal features. In J. Ru. with an apparently normal cardiovascular system the pulmonary blood flow was normal at rest, but the pulmonary arterial pressure was elevated to 19 mm. Hg. This elevation of mean pulmonary arterial

pressure at a level of blood flow through one lung which normally does not cause any pressure elevation suggests a limitation in the expansibility of the vascular bed. Reduction in the number of small vessels and/or a decrease in the cross-section of the lumen of the arterioles and precapillaries are the most likely causes

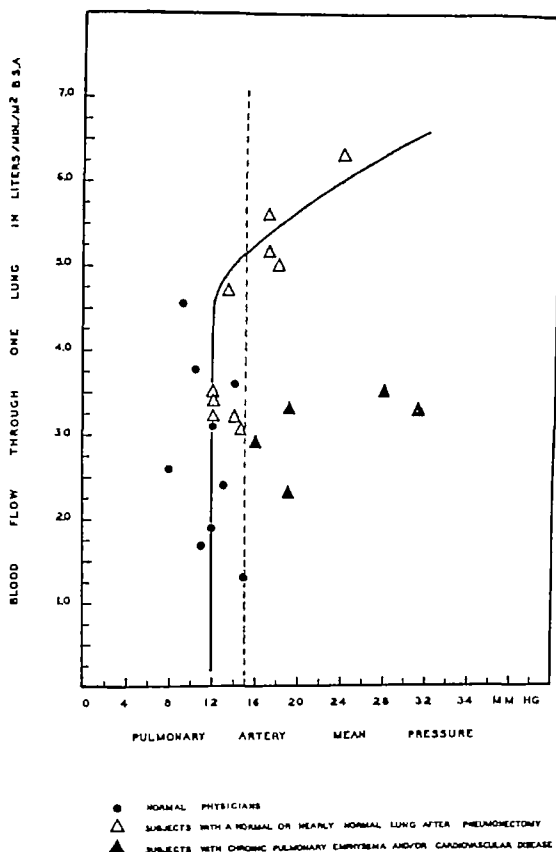


Fig. 9.—Relationship between blood flow through one lung and mean pulmonary arterial pressure in 3 normal subjects and 8 subjects after pneumonectomy.

of this limitation. Since it was demonstrated by the gas tension technique that the blood flow was suppressed in fairly large areas of the lung this finding may be related to the elevated pulmonary artery pressure. In V. Wat., studies of the pulmonary blood flow and pulmonary arterial pressure were made also at

rest and repeated during a steady state of moderate exercise. The pulmonary blood flow at rest was low, and the pulmonary arterial pressure was abnormally elevated to 19 mm. Hg at a level of blood flow through one lung which in normal subjects does not increase pulmonary arterial pressure. During a steady state of moderate exercise the mean pressure in the pulmonary artery rose to the high value of 31 mm. Hg, while the pulmonary blood flow increased to 142 per cent of the resting blood flow. The interpretation of the data is somewhat complicated by the presence in this case of generalized systemic arteriosclerosis associated with systemic hypertension. There is evidence to support the view that the cardiac output is sometimes reduced in systemic hypertension, although clinical and physiologic manifestations of pulmonary venous hypertension may not be present.¹² However, in V. Wat. the exercise tolerance was remarkable and the cardiac output increase during moderate exercise was fairly large, a finding which does not favor the diagnosis of incipient left ventricular insufficiency. Therefore, the possibility of a restriction to the pulmonary vascular bed as a cause of pulmonary hypertension may well be considered also in this case.

In summary, the remaining lung in these 2 cases was the site of chronic emphysematous change of a nonobstructive type. This process originated some time after pneumonectomy, causing alterations in the distribution of alveolar ventilation and perfusion of an unusual character. The pattern of alteration was distinct from that observed in a third patient in whom pneumonectomy was performed in the presence of chronic pulmonary emphysema of the obstructive type. In this case the studies of respiratory gas tensions were not made, but the reduction of the arterial blood oxygen saturation at rest and during the standard exercise test indicates that poorly ventilated alveoli were still perfused. As a result of restriction of the vascular bed in areas of the lung still effectively ventilated, the total ventilation in J. Ru. and V. Wat. was almost double that of normal in all states of physical activity. Whether this constant state of hyperventilation contributed to the maintenance of a large breathing capacity in the presence of a moderate degree of nonobstructive emphysema is a matter of interesting speculation, and is in agreement with similar observations made in an entirely different group of subjects with chronic pulmonary disease.¹³ The study of the dynamics of the pulmonary circulation which revealed a state of mild pulmonary hypertension at rest, more severe in one case during exercise, suggests that the pulmonary vascular bed was limited in its distensibility or restricted in its size. An alternate and/or complementary explanation is offered that in the case with generalized arteriosclerosis and systemic hypertension a latent state of left ventricular insufficiency existed. This would cause an increase in pulmonary venous pressure of which the pulmonary artery pressure would be a reflection.

DISCUSSION

The data presented in this paper will be discussed from 3 different points of view, as they may interest the physiologist, the pathologist, and the chest surgeon.

1. In previous papers dealing with the subject of physiologic readjustments after pneumonectomy several conclusions had been reached. These are well supported by some observations reported here.

(a) The size of the lung in deep inspiration is greater than that of an homologous normal lung, since mediastinal displacement toward the contralateral side is not prevented in the state of maximum inflation by the presence of a contralateral lung.

(b) The reduction of maximum breathing capacity is much less than expected from the volumetric reduction of the chest bellows, unless a significant degree of pulmonary distention develops. Good coordination in the functioning muscles of the chest cage and the absence of any obstruction to air flow help to maintain the efficient utilization of the remaining ventilatory space.

(c) The increase of the ratio effective tidal air/functional residual air, which takes place whenever the total ventilation is performed by a single lung, improves the intrapulmonary mixing of gas.

The quantitative relationships existing between alveolar ventilation and alveolar perfusion in a single lung, early and late, after pneumonectomy have also been studied. It would appear from these studies that in children, young adults, or middle-aged subjects, the adjustments are nearly perfect as long as the remaining lung is free of persisting pathologic change or does not become unduly distended as a result of mediastinal dislocation. However, if such changes are present two types of abnormalities in the distribution of ventilation and blood flow are observed, both characterized by a reduction in the percentage of effective blood flow (perfusion of poorly ventilated alveoli) but associated in the first type only with a reduction in the percentage of alveolar ventilation (ventilation of poorly perfused alveoli). These abnormalities are always slight and related to pulmonary distention and/or discrete pathologic processes. In 2 elderly patients with manifestation of mild nonobstructive emphysema a third type of abnormality was observed, characterized by a reduction in the percentage of the effective alveolar ventilation (ventilation of poorly perfused alveoli), while the percentage of effective pulmonary blood flow (perfusion of well-ventilated alveoli) remained normal. This abnormality is different from that which is observed in patients with bilateral obstructive emphysema where very low effective pulmonary flow and effective alveolar ventilation are found.¹⁴ It suggests a pathologic process restricting the vascular bed alone and creating thereby a need for a considerable increase in total ventilation in order to maintain normal gas exchanges through effective alveolar ventilation of well-perfused pulmonary capillaries. As a result, the breathing reserve is reduced in all states of physical activity with a resultant greater tendency to dyspnea.

Studies of the diffusion of oxygen across the pulmonary membrane indicate that it takes place in a normal manner. If it is postulated that in pneumonectomy the potential number of capillaries is reduced by one-half, the persistence of a normal alveolo-arterial pO_2 gradient at a low level of oxygenation means that the area available for diffusion at rest is nonetheless adequate. The number

of capillaries perfused at rest, therefore, compares favorably with the number of capillaries perfused in both lungs of normal subjects under the same basal conditions. Any pathologic process which encroaches upon the vascular bed of the remaining lung should tend to increase this gradient and, therefore, should tend to decrease the diffusion constant of the lung, defined as the amount of oxygen in cubic centimeters which will diffuse from the alveoli into the blood per minute in response to a mean pressure gradient of 1 mm. Hg.¹⁵ This diffusion constant of the lung, which varies according to the size of the diffusing area, has been calculated in 5 cases, using the graphic method developed by Lillenthal and associates.⁶ Other cases in this series in which the oxygen consumption was too high for useful comparison have been excluded. As seen in Table XVI, the diffusion constant in 3 young subjects with pneumonectomy was remarkably close to the average figure obtained in a group of young adults with two lungs.⁶ In the older subjects, G. H. and V. Wat., aged, respectively, 48 and 72 years, the figures are somewhat lower. In the absence of comparable figures in a group of normal subjects of the same age, it cannot be stated whether or not they are abnormally low. However, they suggest a reduction in the size of the diffusing surface, that is, a reduction in the number of capillaries, which in the case of V. Wat. is consistent with the results of the study of ventilation-perfusion relationships discussed previously.

TABLE XVI. APPROXIMATE DIFFUSION CONSTANT OF THE LUNG IN 5 CASES AFTER PNEUMONECTOMY

SUBJECT	AGE (YR.)	O ₂ CONSUMPTION		TIDAL AIR		ALV.-ART. PO ₂ GRADIENT		MEMBRANE COMPONENT LOW (MM. HG)	MEAN ALV.-CAP. PO ₂ GRADIENT ΔPO ₂ (MM. HG)	DIFFUSION CONSTANT* D O ₂
		HIGH (C.C. PER MIN.)	LOW (C.C. PER MIN.)	HIGH (C.C.)	LOW (C.C.)	HIGH (MM. HG)	LOW (MM. HG)			
Normal (average of 6) †	33	—	258	—	—	9	9.5	8.9	13.3	21
J. Ra.	15	222	234	531	586	11	9	8.2	12	20
J. Lo.	19	254	245	478	412	21	7	5.6	10	25
R. G.	21	254	246	364	303	4	6	5.7	10	25
G. H.	48	247	223	424	474	16	12	10.9	16	14
V. Wat.	72	228	234	390	427	11	11	10.2	15	16

*Calculated from Fig. 5 (in Lillenthal et al.⁶)

†From data collected by Lillenthal et al.⁶; ages ranged from 28 to 36 years.

The observations of the dynamics of the pulmonary circulation provide an unusual opportunity for the study of the deformability of the pulmonary vascular bed at a rate of blood flow through one lung exceeding that observed so far in normal subjects. In studies reported recently⁸ on 3 normal subjects, the mean pulmonary arterial pressure was found not to change as the pulmonary blood flow increased up to nearly 10 liters per minute per m² B.S. during a steady state of severe exercise. The calculated pulmonary vascular resistance was reduced at that level of blood flow to one-third of its value in the resting state. Similar observations have been made independently by Hickam and Cargill.⁷

In Fig. 9 the blood flow through each lung of these normal individuals at various levels of activity is plotted against the mean pulmonary arterial pressure. It is seen that the mean pulmonary arterial pressure remained less than 15 mm. Hg with blood flow through each lung up to 5 liters per minute per m^2 B.S. With a pulmonary blood flow of less than 5 liters per minute per m^2 B.S. the mean pulmonary arterial pressure remained within normal limits in 5 observations made in 5 subjects after pneumonectomy. In 4 of them during moderate exercise, the mean pulmonary arterial pressure rose progressively as the blood flow increased beyond this level. As all these subjects had a normal or nearly normal lung, they revealed a fundamental characteristic of the curve relating blood flow to pressure in the pulmonary vascular bed by reaching a rate of blood flow through one lung exceeding that observed in normal individuals. The distensibility curve of the pulmonary vascular bed, which is straight and vertical up to a blood flow of 5 liters per minute per m^2 B.S. shows a definite inflection at this level. The characteristics of this curve are of potential interest in the interpretation of the possible causes of pulmonary hypertension in congenital defects of the heart and of the large vessels. If the same curve applies to very young children as well as to adults, it would seem that a moderate increase in the mean pulmonary arterial pressure, with a blood flow of less than 5 liters per minute per m^2 B.S. through each lung is due either to pulmonary vascular changes or to pulmonary venous hypertension. Above this level of flow, an additional factor must be considered, as a large blood flow by itself may cause pulmonary hypertension.

In the remaining 5 observations, plotted in Fig. 9, the pressure in the pulmonary artery was abnormally high in the resting state and increased further during exercise, while the blood flow was significantly less than 5 liters per minute per m^2 B.S. The probable causes of such pulmonary hypertension have been discussed already.

2. The final word is left to the pathologist regarding the problems of pulmonary hypertrophy in young subjects, and of the exact nature of the pathologic processes observed in the elderly subjects. The data in the children (H. W. and R. G.) who have grown to adult age with a single lung do not contradict the concept of pulmonary hypertrophy. Physiologically their lungs are perfect. Moreover, the remarkable efficiency of their lungs and that of an adult (J. Rus.) 41 years of age at the time of pneumonectomy may well be explained without recourse to this concept. The question remains unanswered as to whether the physiologic pattern presented by the two elderly subjects is related to a particular type of degenerative change occurring in the lungs of older persons after pneumonectomy, or whether they developed coincidentally a mild state of senile emphysema, as seems not improbable at their age.

3. The data presented are of more than academic interest to the thoracic surgeon. It has been shown in 2 young subjects and in one middle-aged subject that pulmonary distention following pneumonectomy does not inevitably develop in the absence of thoracoplasty. Conversely, it has been shown that thoracoplasty complicated by scoliosis may actually cause pulmonary distention. The question of optional or systematic thoracoplasty following pneumonectomy for

TABLE XVII. INFLUENCE OF THORACOPLASTY UPON DYSPNEA IN 31 CASES AFTER PNEUMONECTOMY

DIAGNOSIS	NO. OF CASES	AGE (YR.)	PNEUMONECTOMY WITHOUT THORACOPLASTY				PNEUMONECTOMY WITH THORACOPLASTY			
			NO PULMONARY DISTENTION		PULMONARY DISTENTION		NO PULMONARY DISTENTION		PULMONARY DISTENTION	
			NO. OF CASES	RESULT*	NO. OF CASES	RESULT*	NO. OF CASES	RESULT*	NO. OF CASES	RESULT*
Carcinoma of the bronchus										
Short survival (mo.)	3	39	2	—	1	—				
Long survival (yr.)	4	59			3	Good, 2 Fair, 1			1	Good, 1
Chronic pulmonary suppuration										
Children	4	12	2	Good, 2	1	Good, 1			1	Good, 1
Adults	13	39	4	Good, 4	3	Fair, 2	3	Good, 3	2	Poor, 2
Tuberculosis bronchus	7	34	1	Good, 1	3	Poor, 1 Poor, 3	1	Good, 1	2	Poor, 2
Recapitulation	31		9	Good, 7 Not estimated, 2	11	Good, 3 Fair, 3 Poor, 4 Not estimated, 1	4	Good, 4	6	Good, 2 Poor, 4

*For interpretation see text.

chronic suppuration of the lung and carcinoma of the lung has not yet been solved for lack of adequate statistics. Table XVII has been prepared in an attempt to provide such statistical data. They refer to repeated measurements of lung volume, of maximum breathing capacity, and of ventilation during exercise and recovery made in 31 patients a short time after pneumonectomy. Whenever 2 successive measurements after a six-week interval showed an increase in residual air and a reduction in maximum breathing capacity, thoracoplasty was recommended. The thoracoplasty was performed as a matter of routine in a few cases, or not performed when recommended in a few others. Estimation of the final functional result was based on the presence and the degree of shortness of breath during the recovery period of the standard exercise test.

The results may be summarized as follows:

- In 13 cases without thoracoplasty and pulmonary distention the final results were good in 9, fair in 1, and in 2 patients the survival period after pneumonectomy was too short to permit a proper evaluation of the result.
- In 11 cases without thoracoplasty but with pulmonary distention the results were good in 3, fair in 3, poor in 5. In the category of poor results are included 2 patients with tuberculosis of the remaining main bronchus.

- (c) In 4 cases thoracoplasty was performed without evidence of pulmonary distention and the final results were good in all of them.
- (d) Finally, in 6 cases thoracoplasty was performed to control progressive pulmonary distention, and the results were good in 2 and poor in 4. Among these 4 cases are 2 patients reported in this paper who developed scoliosis and 2 patients in whom pulmonary resection was performed for bronchial tuberculosis complicated by fibrothorax of long standing.

In conclusion it can be said that :

1. In the absence of thoracoplasty pneumonectomy is not inevitably followed by evidence of distention of the remaining lung.
2. If pneumonectomy is complicated by pulmonary distention, thoracoplasty is advisable, but the final result depends to a great extent upon the presence or absence of a pathologic process within the remaining lung.
3. Thoracoplasty may cause scoliosis and be followed by poor results due to pulmonary distention.

The observations reported in this paper concerning the dynamics of the circulation demonstrate that a state of mild pulmonary hypertension inevitably develops in the course of mild exercise, when the entire right ventricular output, circulating through a single lung, reaches an easily attainable level. In the course of strenuous activity, the blood flow in a single lung may reach very high values, which, in accordance with the general configuration of the curve relating blood flow and pressure in the pulmonary vascular bed, may result in a state of very marked pulmonary hypertension. The inference could be made from these observations that physical activity should be restricted after pneumonectomy. However, from the results of electrocardiographic studies with unipolar precordial leads¹⁶ it seems reasonable to state that the mass of the right ventricle in the patients of this series was not increased and that therefore right ventricular hypertrophy had not developed after several years, although the subjects followed had not been restricted in their physical activity.

SUMMARY

1. The adaptation to pneumonectomy of the ventilation, the respiratory gas exchange, and the pulmonary circulation has been investigated in 16 individuals.
2. The methods of investigation used include the measurements of pulmonary blood flow and pressure, and an analysis of the alveolar ventilation-perfusion relationships, in addition to the measurements employed in previously reported studies.
3. The results are discussed in relation to age, time of study after pneumonectomy, development of pulmonary distention, and presence or absence of thoracoplasty.
4. Examination of pulmonary circulation under varying conditions of activity after pneumonectomy provides a unique opportunity to observe certain properties of the pulmonary vascular bed.

The authors acknowledge their indebtedness to Dr. F. Berry, Dr. J. Burns Amberson, and Dr. D. W. Richards, Jr., for their continuous support and interest, and to Mrs. Marianne S. Lester, Miss Blanche Spierto, and Miss Dorothy Allen for their technical help.

REFERENCES

1. Lester, C. W., Cournand, A., and Riley, R. L.: Pulmonary Function After Pneumonec-tomy in Children, *J. THORACIC SURG.* 11: 529, 1942.
2. Cournand, A., Himmelstein, A., Riley, R. L., and Lester, C. W.: A Follow-Up Study of the Cardiopulmonary Function in Four Young Individuals After Pneumonec-tomy, *J. THORACIC SURG.* 16: 30, 1947.
3. Cournand, A., and Berry, Frank B.: The Effect of Pneumonec-tomy Upon Cardiopulmo-nary Function in Adult Patients, *Ann. Surg.* 116: 532, 1942.
4. Birath, G.: Lung Volume and Ventilation Efficiency, *Acta Med. Scandinav. Supp.* 154, pp. 1-215, 1944.
5. Riley, R. L., Proemmel, D. D., and Franke, R. E.: A Direct Method for Determination of Oxygen and Carbon Dioxide Tensions in Blood, *J. Biol. Chem.* 161: 621, 1945.
6. Lillenthal, J. L., Jr., Riley, R. L., Proemmel, D. D., and Franke, R. E.: An Experi-mental Analysis in Man of the Oxygen Pressure Gradient From Alveolar Air to Arterial Blood During Rest and Exercise at Sea Level and at Altitude, *Am. J. Physiol.* 147: 199, 1945.
7. Hickam, J. B., and Cargill, W. H.: Effect of Exercise on Cardiac Output and Pulmo-nary Arterial Pressure in Normal Persons and in Patients With Cardiovascular Disease and Pulmonary Emphysema, *J. Clin. Investigation* 27: 10, 1948.
8. Riley, R. L., Himmelstein, A., Motley, H. L., Weiner, H. M., and Cournand, A.: Studies of Pulmonary Circulation at Rest and During Exercise in Normal Individuals and in Patients With Chronic Pulmonary Disease, *Am. J. Physiol.* 152: 372, 1948.
9. Dexter, L., Haynes, F. W., Burwell, C. S., Eppinger, E. C., Sosman, M. C., and Evans, J. M.: Studies of Congenital Heart Disease. II. The Pressure and Oxygen Con-tent of Blood in the Right Auricle, Right Ventricle, and Pulmonary Artery in Control Patients, With Observations on the Oxygen Saturation and Source of Pulmonary "Capillary" Blood, *J. Clin. Investigation* 26: 554, 1947.
10. Riley, R. L., and Cournand, A.: "Ideal" Alveolar Air and the Analysis of Ventilation-Perfusion Relationships in the Lungs, *J. A. Phys.* 1: 825, 1949.
11. Baldwin, E. deF., Cournand, A., and Richards, D. W., Jr.: Pulmonary Insufficiency. I. Physiological Classification, Clinical Methods of Analysis, Standard Values in Normal Subjects, *Medicine* 27: 243, 1948.
12. Ferrer, M. L., Harvey, R. M., Richards, D. W., Jr., and Cournand, A.: Unpublished data.
13. Baldwin, E. deF., Cournand, A., and Richards, D. W., Jr.: Pulmonary Insufficiency. II. A Study of Thirty-nine Cases of Pulmonary Fibrosis, *Medicine* 28: 201, 1949.
14. Riley, R. L., Austrian, R., Donald, K. W., and Cournand, A.: The Relation of Effective Pulmonary Blood Flow to Total Pulmonary Blood Flow in Normal Man and in Patients With Various Types of Chronic Pulmonary Disease, *Proc. Am. Soc. Clin. Investigation*, 1949. (In press.)
15. Krogh, M.: The Diffusion of Gases Through the Lungs of Man, *J. Physiol.* 49: 271, 1914.
16. Johnson, John B.: Personal communication.

FIBROSARCOMA OF THE BRONCHUS

REPORT OF A CASE DIAGNOSED BY BRONCHOSCOPY AND TREATED BY PNEUMONECTOMY

JAMES CARSWELL, JR., M.D., AND NELSON H. KRAEFT, M.D.
McKINNEY, TEXAS

PRIMARY fibrosarcoma of the lung is stated to be among the rarest of tumors.^{4, 11} In earlier reports one finds the term "primary sarcoma of the lung" often without further identification as to cell type. It is apparent that the term excludes lymphosarcomas since these are primarily extrapulmonary, arising in bronchial or mediastinal lymph nodes.⁷ Ball¹ in 1931 reported a fibrosarcoma primary in and replacing the left lower lobe of the lung. He reviewed the literature from 1900 to 1931 and found only thirteen well-documented cases of primary sarcoma of the lung. These included two fibrosarcomas, three spindle-cell sarcomas, six round-cell sarcomas, one "plexiform" sarcoma, and one fibrochondroadenoma with sarcomatous changes. Rosenblum and Gasul¹¹ in 1931 found thirteen cases of primary sarcoma of the lung reported in the pediatric literature. These included one round-cell sarcoma and one spindle-cell sarcoma; the cell type of the others was not mentioned. They added one case of their own, designated only as primary sarcoma of the lung in an infant. Of the cases collected by Rosenblum and Gasul, three had been included in the group collected by Ball.

Ellis⁶ reported one case of spindle-cell sarcoma of the lung in a series of 7,272 autopsies. In the report of Massachusetts General Hospital Case 22441¹⁴ Mallory discussed the first primary fibrosarcoma of the lung in a series of 8,000 autopsies. Three other cases have since been reported from this hospital.^{15, 16, 17} Oelsner and associates,⁸ in a recent report on 412 patients with primary cancer of the lung covering a period of twelve years, included two cases of fibrosarcoma in the group treated by resection; whether or not these were primary in a bronchus or diagnosed bronchoscopically was not stated in this report.

Unlike other tumors of the lung, sarcomas of any cell type have been diagnosed bronchoscopically only on rare occasions. Kahler is quoted by Pollak and associates¹⁰ as making the earliest reference to bronchial sarcoma. In 1911 he reported a patient having a mass completely obstructing the right bronchus. A piece of the tissue coughed up was identified as myxosarcoma. Pollak and his co-workers reported a fibrosarcoma of the bronchus in a 19-year-old white man. Baum and associates² in 1939 reported a spindle-cell sarcoma of the bronchus in a 29-year-old white woman. In a review of the literature they were able to find only one such case previously diagnosed bronchoscopically.

From the Veterans Administration Hospital.

Received for publication Sept. 27, 1948.

Sponsored by the Veterans Administration and published with the approval of the Chief Medical Director. The statements and conclusions published by the authors are the result of their own study and do not necessarily reflect the opinion or policy of the Veterans Administration.

This was the case of Herrnheiser reported in 1935 (quoted by Baum²). Massachusetts General Hospital Case 24202¹⁵ was a 13-year-old girl with fibrosarcoma of the bronchus diagnosed bronchoscopically. Vinson¹³ has reported a lymphosarcoma of the bronchus, but this type of tumor is not included in most reviews of the subject for reasons already noted.

Ewing⁷ recognized diffuse spindle-cell, small round-cell, and large round-cell sarcomas of the lung, in addition to the lymphosarcomas which form a separate group. The possibility of mistaking reparative or inflammatory tissue for round-cell or spindle-cell sarcoma was emphasized by Ewing⁷ and Boyd.⁵ It is thought that round-cell or anaplastic carcinoma of the bronchus was often reported as sarcoma in the earlier literature.^{5, 9} Ball,¹ after excluding all cases in which the diagnosis was inadequately supported, found that primary sarcoma of the lung could be divided into two groups. The first is the diffuse or circumscribed tumor of slow progression occurring in adults and presenting, microscopically, various stages of differentiation of fibrosarcoma. The second group occurs in young individuals, is more rapidly progressive, is composed of "round" cells, and is subject to more necrosis since it is more cellular. Steward¹² has reported one case in which a mixed sarcoma occurred in the left lung of a 12-year-old girl. This tumor had some areas of spindle-cell predominance and other areas in which round cells were more numerous. No case of round-cell sarcoma diagnosed bronchoscopically has been found, probably because this tumor occurs in younger individuals and is more rapidly progressive than fibrosarcoma.

Baum and associates^{2, 3} treated their patient by partial bronchoscopic removal followed by x-ray therapy. This therapy was given over a period of two years as dictated by local recurrence found bronchoscopically on two occasions. The interval between the two x-ray treatments was about twenty months. Death occurred thirty-two months after diagnosis, due to rapidly progressive extensive bilateral pulmonary tuberculosis. It was thought that the activity and caseation of the tuberculous lesions were accelerated by the heavy dosage of roentgen rays. One patient (Massachusetts General Hospital Case 24202¹⁵) died of massive hemorrhage from the base of the tumor during attempted pneumonectomy. The tumor caused complete obstruction of the right bronchus and was apparently detached from its base during attempt to deflate the distended, obstructed lung. Churchill, in discussing the case, pointed out the advisability of clearing the bronchus of tumor by local bronchoscopic removal preoperatively, thus making the lung more manageable at operation.

The patient reported by Pollak and associates¹⁰ was treated by local removal of the tumor bronchoscopically, plus fulguration of its base. This sarcoma was considered on microscopic examination as "locally malignant and if properly removed should not recur." The resemblance to a sarcomatous nasal polyp was mentioned. Treatment was instituted August, 1936. The patient was well and working as a carpenter when last seen in March, 1939, having only minimal symptoms from bronchiectasis which developed distal to the tumor.

CASE REPORT

Illness at Time of Admission.—A 29-year-old college student was admitted to this hospital Dec. 23, 1947, because of severe pain in the left side of the chest, dyspnea, cough, and wheezing. He had lost 23 pounds in the past five months. Symptoms began in July, 1946, with mild dyspnea on exertion, becoming progressively worse until September, when wheezing and occasional small hemoptyses were noted. Cough was mild and not productive except when hemoptysis occurred. Bronchoscopy at this time was reported as negative. Ten days prior to admission the patient developed chills and fever, with severe pain in the left side of the chest. Bronchoscopy was repeated but nothing was found, and he was transferred to this hospital.

Physical Examination.—Physical examination revealed a well-developed, undernourished white man, not acutely ill. He was slightly dyspneic. Skin was pale and showed some evidence of recent weight loss.

Examination of the chest revealed significant flattening of the lower half of the chest on the left, with a marked lag on deep inspiration. Tactile fremitus was diminished in the upper half and absent in the lower half of the left side of the chest. There was dullness to percussion over the lower half on the left, with diminished to absent breath sounds over the entire left side. A definite wheeze was heard over the left lower lung field on deep inspiration. There was a shift to the left of the lower mediastinum. There were no abnormal findings over the right side of the chest. The heart was normal. The remainder of the physical examination was essentially negative.

Röntgenogram of the chest revealed pleural thickening with subpleural fibrosis over the right apex, and atelectasis of the left lower lobe.

Serum examinations for syphilis were negative. Blood and urine analyses revealed no abnormalities. Sputum examinations were repeatedly negative for acid-fast bacilli. Blood chemistry was normal.

Bronchoscopy done Dec. 29, 1947, showed the left main stem bronchus almost completely occluded by a round, smooth, shining, nonulcerated, dark red, fairly firm tumor, attached to the medial wall of the bronchus about $\frac{1}{2}$ cm. distal to the presenting edge of the carina. A portion of the tumor, sufficiently large to establish a free airway, was removed for biopsy without excessive bleeding. The trachea and right bronchial tree were normal.

Pathologic Report.—The pathologist* gave the following report on the bronchial biopsy. Microscopic examination of tissue obtained by bronchial biopsy revealed a small portion of the specimen to be covered by normal-appearing bronchial mucosa, while in another area the mucosa had undergone a pronounced squamous-cell metaplasia. Immediately beneath, but not involving the mucosa, was a pleomorphic cellular neoplasm composed of loosely arranged, spindle-shaped fibroblasts. The fibroblasts possessed delicate cytoplasmic processes and, with phosphotungstic acid hematoxylin stain, demonstrable fibroglia fibrils. The cells were separated by delicate strands of collagen. These cells in occasional areas were arranged in loose nests, vaguely suggestive of whorls, while in most areas the cells remained more discrete with no indication of pattern arrangement. While the cells possessed large hyperchromatic nuclei with a reticular meshwork of nuclear chromatin, there was no indication of mitotic activity. The neoplasm had a rich vascular supply, but in no area could invasion of vessels be demonstrated. Diagnosis was fibrosarcoma of bronchus.

Following bronchoscopic removal of a portion of the tumor for biopsy, the lower lobe atelectasis cleared almost entirely and the mediastinal silhouette returned to normal position.

On Jan. 14, 1948, a left pneumonectomy was done, with division of the main bronchus at the level of the carina. The individual ligation technique was used. Except for a small, firm, discrete subpleural nodule in the mid-portion of the lateral aspect of the upper lobe, the lung presented no gross abnormality. There were no pleural adhesions. There were no grossly enlarged or abnormal hilar nodes encountered. One slightly enlarged node was found in the angle at the bifurcation of the trachea.

*Dr. O. J. Wallenman, Jr.

Gross Findings.—The gross specimen consisted of a collapsed left lung weighing 250 grams and all of the left main bronchus. At a point 5 mm. from the carina the essential findings were small, granular, hemorrhagic lesions of the bronchial mucosa. The mucosa proximal to and distal to the tumor, as well as throughout the entire bronchial tree, was intact and glistening gray. No peribronchial masses were present. The lung parenchyma was not remarkable except for a solitary, subpleural 2 mm. nodule situated in the lateral aspect of the mid-portion of the upper lobe. The hilar lymph nodes were not enlarged and on section did not appear remarkable. A separate node removed from the bifurcation of the trachea measured approximately 7 by 4 by 4 mm. and on section had a minute, central, caseous area surrounded by normal-appearing lymph node.

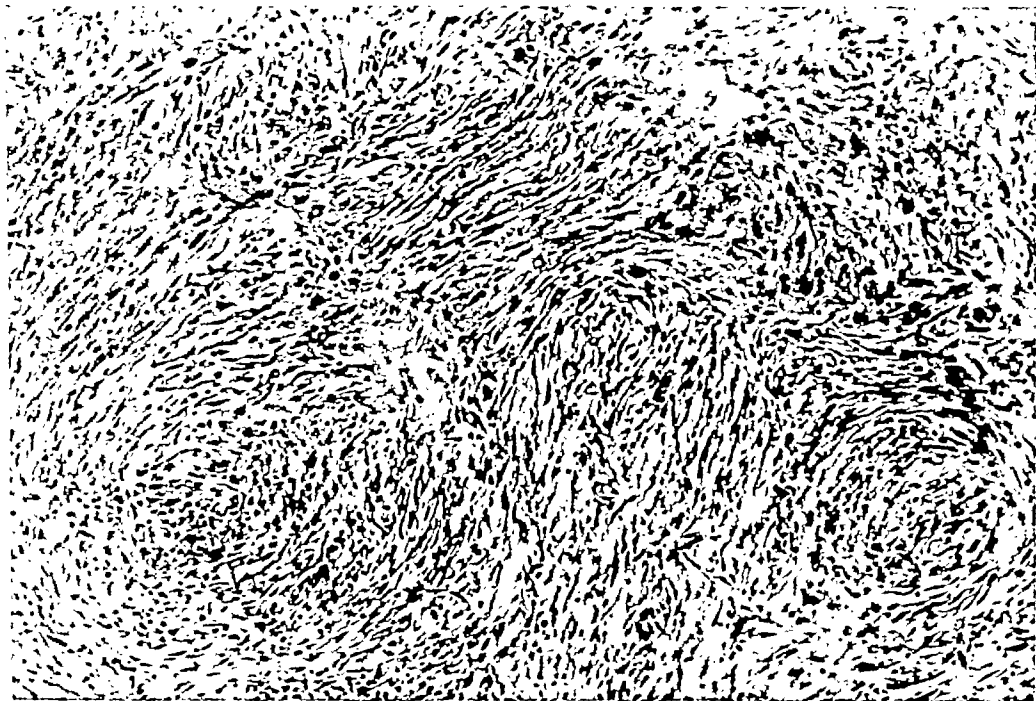


Fig. 1.—Bronchial biopsy showing whorling and interlacing arrangement of fibroblasts. (Hematoxylin and eosin stain, magnification $\times 180$.)

Microscopic Findings.—Microscopic examination of sections removed from the granular portion of the left main bronchus described in the gross findings revealed a polypoid mass covered with metaplastic bronchial mucosa projecting into the lumen of the bronchus. Immediately beneath the mucosa were loosely arranged spindle cells separated by strands of collagen and what appeared to be edema fluid. The spindle cells became more numerous and compact at the base of the polyp, and in this area and deeper in the submucosa and muscularis of the bronchus, the cells tended to lose their spindle shape characteristic; the nuclei were much larger, more hyperchromatic, and in some areas the cells appeared oval to round with the nucleus occupying three-fourths of the cell structure. This more anaplastic appearance was a definite gradient from the more mature fibroblasts comprising the polyp. Although the cells appeared anaplastic and less differentiated than seen in the biopsy specimen, mitotic activity was found with extreme difficulty. In no area was there attempted differentiation into the glandular structures, sheets of cells, or strands reminiscent of bronchial mucosa origin. The tumor was richly vascularized, and in a few areas chronic inflammatory cells were seen, as well as an occasional focus of lipid laden macrophages (Fig. 1). Microscopic examination of the nodule described in the gross findings as situated

beneath the pleura revealed this to be an encapsulated benign chondroma situated adjacent to a small bronchiole in the lung parenchyma. This chondroma was surrounded by a moderately dense fibrous tissue capsule and was composed of mature hyaline cartilage. The adjacent lung parenchyma, as well as multiple sections from various portions of both lobes, showed partial atelectasis without inflammatory reaction. There was no evidence of neoplastic tissue in the lung parenchyma. Microscopic examination of the accompanying lymph node described in the gross findings revealed an encapsulated eosinophilic amorphous material occupying the central portion of the node. The capsule was composed of dense, fibrocollagenous tissue without evidence of cellular inflammatory reaction. Microscopic examination of other lymph nodes removed from the hilus revealed heavy carbon pigmentation, but otherwise were not significantly altered. Diagnoses: (1) Fibrosarcoma, left main bronchus; (2) chondroma, benign; (3) tuberculous lymphadenitis, encapsulated.

The patient had an uneventful postoperative course, and immediately prior to discharge from the hospital on Feb. 3, 1948, bronchoscopy revealed a well-healed suture line on a plane with the carina and the left wall of the trachea. The mucosa appeared normal. Follow-up examination on April 5, 1948, and again on Sept. 1, 1948, seven and one-half months after resection, showed a normal bronchial mucosa. The patient had no complaints, was feeling well and attending school.

COMMENTS

It is apparent, in the light of the infrequency with which primary sarcoma of the bronchus is encountered, that no single plan of treatment can be formulated on the basis of experience. The spindle-cell sarcoma reported by Baum and associates was treated by partial bronchoscopic removal followed by x-ray therapy. Death occurred thirty-two months later from extensive bilateral pulmonary tuberculosis. Local recurrence was observed on two occasions prior to death. This case would suggest that x-ray therapy could present an unwarranted risk in any patient in whom treatment by resection is not contraindicated. The patient with fibrosarcoma of the bronchus discussed by Mallory died of hemorrhage from the base of the tumor during attempt at pneumonectomy. The fibrosarcoma reported by Pollak and associates was treated by local removal and fulguration of the base. They reported that the patient was well three years later.

Our decision to elect pneumonectomy as the treatment of choice in the case reported here was based upon uncertainty as to the depth of invasion of the bronchial wall, although no extrabronchial extension could be demonstrated roentgenologically. A free airway was established at the time of partial bronchoscopic removal and persisted throughout the resection. Subsequent microscopic examination of the base of the tumor showed extension of the neoplasm between the cartilaginous rings to involve the entire thickness of the bronchial wall. Local removal and destruction by fulguration of anything less than the full thickness of the bronchial wall in this case would have failed to eradicate the tumor. Since it is conceivable that fibrosarcoma may simulate bronchial adenoma in its manner of growth, that is, by assuming a "dumb-bell" shape, we feel that pulmonary resection should be carried out proximal to the site of the tumor. The amount of pulmonary tissue to be sacrificed would then depend upon the location of the neoplasm.

SUMMARY

1. Primary sarcoma of the lung is in itself a rare disease, and primary involvement of a bronchus is exceptionally so.

2. A brief review of the American literature indicates that a total of thirty-one cases of primary sarcoma of the lung have been reported. We are able to find only six reported cases of sarcoma primary in the bronchus. The treatment of these has not been uniform.

3. The treatment of primary sarcoma of the bronchus is discussed.

4. A case of primary fibrosarcoma of the bronchus, diagnosed bronchoscopically and treated by pneumonectomy is presented in detail.

REFERENCES

1. Ball, H. A.: Primary Pulmonary Sarcoma; Review With Report of Additional Case, *Am. J. Cancer (Supp.)* 15: 2319, 1931.
2. Baum, Otto S., Richards, John H., and Ryan, Maxwell D.: A Case of Atelectasis of Right Middle and Lower Lobes With Bronchoscopy Demonstrating Spindle Cell Sarcoma of Right Main Bronchus, *Ann. Int. Med.* 12: 699, 1938.
3. Baum, Otto S., Silverman, Gertrude, Rosh, Rieva, and Riley, Richard: A Case of Spindle Cell Sarcoma of the Bronchus. Supplementary Report, *Ann. Int. Med.* 17: 750, 1942.
4. Boyd, William: *Surgical Pathology*, Philadelphia, 1947, W. B. Saunders Company.
5. Boyd, William: *Pathology of Internal Diseases*, Philadelphia, 1944, Lea & Febiger.
6. Ellis, R. C.: Primary Sarcoma of Lung With Brain Metastases, *J. Kansas M. Soc.* 40: 243, 1939.
7. Ewing, J.: *Neoplastic Diseases*, Philadelphia, 1942, W. B. Saunders Company.
8. Ochsner, A. L., DeBakey, Michael, and Dixon, J. Leonard: Primary Cancer of Lung, *J. A. M. A.* 135: 321, 1947.
9. Pilot, I.: Mesenchymatous Tumors of Lung and Pleura, *Radiology* 14: 391-400, 1930.
10. Pollak, B. S., Cohen, S., Barrone, M. G., and Gnassi, A. N.: Primary Sarcoma of Bronchus, *Am. J. Roentgenol.* 41: 909, 1941.
11. Rosenblum, P., and Gasul, B. M.: A Case of Primary Sarcoma of the Lung in an Infant 29 Months of Age, *Arch. Pediat.* 48: 63, 1931.
12. Steward, W. Dean: Sarcoma of Lung, *Am. Rev. Tuberc.* 40: 224, 1939.
13. Vinson, P.: Primary Malignant Disease of Tracheobronchial Tree, *J. A. M. A.* 107: 258, 1936.
14. Massachusetts General Hospital Case 22441: *New England J. Med.* 215: 837, 1936.
15. Massachusetts General Hospital Case 24202: *New England J. Med.* 218: 843, 1938.
16. Massachusetts General Hospital Case 24472: *New England J. Med.* 219: 854, 1938.
17. Massachusetts General Hospital Case 31271: *New England J. Med.* 233: 18, 1945.

FIBROSARCOMA OF THE BRONCHUS

HARRISON BLACK, M.D.*

St. Louis, Mo.

DESPITE the extensive experience with pulmonary neoplasms in the past fifteen years and despite the large series of cases that have been reported from various clinics, primary sarcoma of the lung in general and primary intra-bronchial fibrosarcoma in particular must still be regarded as lesions of considerable rarity. At the turn of the century Seydel¹ reviewed a large number of cases reported up to that time. Among these were many "round-cell sarcomas" (probably mostly undifferentiated bronchial carcinomas or lymphosarcomas of the mediastinum with extension to the lung). There were also several "spindle-cell sarcomas" mentioned, but we have been unable to verify any of them as primary fibrosarcomas of the bronchus from the data available in the reports. In this connection it is well to recall Stout's²³ statement that "it is an utter waste of paper to report a tumor by this title [spindle cell sarcoma] unless the description and illustrations are so good that an expert oncologist, aided by the clinical history, can recognize the true nature of the growth from them."

A similar lack of adequate description makes it impossible to confirm as primary bronchial fibrosarcoma a single one of the cases of sarcoma of the lung which Adler¹ listed in his book published in 1912. Moreover, Adler stated that he had never seen a primary sarcoma of the lung in his personal experience and called attention to the difficulty often encountered in distinguishing very undifferentiated carcinomas of the lung (including the so-called "oat-cell" tumor) from sarcomas. Because of the failure of many authors to make this important differentiation, their reported cases have had to be discarded as unsubstantiated.

Edwards⁶ noted that in the late 1920's there was a rapid disappearance from the literature of reports of large numbers of cases of primary sarcoma of the lung because of Bernard's reiteration that a more careful study would separate the very undifferentiated epithelial tumors from the sarcomas. Many reports are still encountered, however, in which the criteria for establishing the diagnosis of sarcoma are incompletely described.

Stout²³ has done much to clarify the confused subject of fibrosarcoma, and it would be well to set down his criteria for this diagnosis before evaluating the individual case reports. All tumors except those composed of fibroblasts which produce collagen and reticulin fibers (the latter being wrapped around the individual tumor cells in a characteristic manner) have been excluded in this report. The variation in degree of production of these fibers and the lack of collagen in the more undifferentiated fibrosarcomas are pointed out. The parent cells are almost always fusiform when cut along the long axis and are dis-

From the Laboratory of Surgical Pathology, Department of Surgery, Washington University School of Medicine, and Barnes Hospital.

Received for publication Jan. 15, 1919.

*Fellow in Chest Surgery.

SUMMARY

1. Primary sarcoma of the lung is in itself a rare disease, and primary involvement of a bronchus is exceptionally so.

2. A brief review of the American literature indicates that a total of thirty-one cases of primary sarcoma of the lung have been reported. We are able to find only six reported cases of sarcoma primary in the bronchus. The treatment of these has not been uniform.

3. The treatment of primary sarcoma of the bronchus is discussed.

4. A case of primary fibrosarcoma of the bronchus, diagnosed bronchoscopically and treated by pneumonectomy is presented in detail.

REFERENCES

1. Ball, H. A.: Primary Pulmonary Sarcoma; Review With Report of Additional Case, *Am. J. Cancer (Supp.)* 15: 2319, 1931.
2. Baum, Otto S., Richards, John H., and Ryan, Maxwell D.: A Case of Atelectasis of Right Middle and Lower Lobes With Bronchoscopy Demonstrating Spindle Cell Sarcoma of Right Main Bronchus, *Ann. Int. Med.* 12: 699, 1938.
3. Baum, Otto S., Silverman, Gertrude, Rosh, Rieva, and Riley, Richard: A Case of Spindle Cell Sarcoma of the Bronchus. Supplementary Report, *Ann. Int. Med.* 17: 750, 1942.
4. Boyd, William: *Surgical Pathology*, Philadelphia, 1947, W. B. Saunders Company.
5. Boyd, William: *Pathology of Internal Diseases*, Philadelphia, 1944, Lea & Febiger.
6. Ellis, R. C.: Primary Sarcoma of Lung With Brain Metastases, *J. Kansas M. Soc.* 40: 243, 1939.
7. Ewing, J.: *Neoplastic Diseases*, Philadelphia, 1942, W. B. Saunders Company.
8. Ochsner, A. L., DeBaKey, Michael, and Dixon, J. Leonard: Primary Cancer of Lung, *J. A. M. A.* 135: 321, 1947.
9. Pilot, I.: Mesenchymatous Tumors of Lung and Pleura, *Radiology* 14: 391-400, 1930.
10. Pollak, B. S., Cohen, S., Barrone, M. G., and Gnassi, A. N.: Primary Sarcoma of Bronchus, *Am. J. Roentgenol.* 41: 900, 1941.
11. Rosenblum, P., and Gasul, B. M.: A Case of Primary Sarcoma of the Lung in an Infant 29 Months of Age, *Arch. Pediat.* 48: 63, 1931.
12. Steward, W. Dean: Sarcoma of Lung, *Am. Rev. Tuberc.* 40: 224, 1939.
13. Vinson, P.: Primary Malignant Disease of Tracheobronchial Tree, *J. A. M. A.* 107: 258, 1936.
14. Massachusetts General Hospital Case 22441: *New England J. Med.* 215: 837, 1936.
15. Massachusetts General Hospital Case 24202: *New England J. Med.* 218: 843, 1938.
16. Massachusetts General Hospital Case 24472: *New England J. Med.* 219: 854, 1938.
17. Massachusetts General Hospital Case 31271: *New England J. Med.* 233: 18, 1945.

after pneumonectomy for a neoplasm that is not described except by the general term sarcoma. In another larger series of cases Jackson¹¹ mentioned six sarcomas which were all seen as endobronchial growths and diagnosed by bronchoscopic biopsy. Only one of these was a "spindle-cell sarcoma," and there was no further information as to treatment, follow-up, or postmortem. As no photomicrographs or histologic description were available this case has also been excluded.

In recent years the most comprehensive paper on the subject of primary pulmonary sarcoma is that of Ball.² In his review of the literature he found fourteen cases which he considered verifiable in addition to his own. Among these were six cases diagnosed as "round-cell sarcoma," a term which probably implies that they were lymphosarcomas. Since this neoplasm is not being considered in this report, and since they cannot be classified more accurately from the published data, they have been eliminated. Five others, including those of Edwards,¹ Stevens,²¹ and Pritchard²⁷ were found to have inadequate histologic study and description. Another was termed a "fibrochondroadenoma with osteoid tissue." This leaves only the cases of Boschowsky⁶ and Ball,² both of which have adequate histologic description to substantiate the diagnosis of fibrosarcoma. These tumors, however, occupied almost the entire lung and their site of origin, whether from the pleura or pulmonary parenchyma, is not clear. From the descriptions it is impossible to include them with polypoid fibrosarcomas of the bronchus. Other cases which were discarded for the same reasons are those of Ellis,⁹ Mallory,^{18, 21} Lyssunkin,^{16, 17} Scheidegger,²⁰ Stohr and Sachs,²² and Klemperer and Rabin¹³ although they are well-documented fibrosarcomas of the lung or pleura.

Other reports of tumors which were diagnosed as fibrosarcoma primary in the lung have been reviewed but have been found deficient in one or more respects for inclusion with verified cases. Thus, the reports of Rolleston and Trevor²⁴ and Lange¹⁴ must be discarded because of inadequate histologic description and that of Mallory²⁰ because the pulmonary tumor was not definitely proved as primary, although the histologic description appears to be adequate.

This leaves only five cases from the entire medical literature (see Table I) which can be considered primary fibrosarcomas of the bronchus. All are polypoid bronchial tumors with varying extension into the lung parenchyma and have either good histologic descriptions or photomicrographs from which it is possible to verify the diagnosis.

CASE 1 (Baum^{2, 4}).—The tumor was removed through the bronchoscope followed by x-ray therapy. The author stated that there was no evidence of persistent tumor at the autopsy of this patient who died of tuberculosis two years and nine months after the onset of illness, but one wonders whether the "bundles of apparently fibrous tissue running in various directions and abutting against necrotic tuberculous tissue" found at the site of the original tumor do not represent persistence. It would be unusual indeed in view of Stout's experience²³ to find sterilization of a fibrosarcoma by x-ray therapy.

CASE 2 (Johns and Sharpe¹²).—No treatment was given, and the patient died of respiratory complications fifteen months after the onset of symptoms.

tributed in bundles or cords which interlace in gentle curves. Occasionally areas are found in which the bundles abruptly change direction giving the appearance of whorls. Stout thus includes under the heading of fibrosarcoma those tumors which others have termed "neurofibrosarcoma," and to this classification we emphatically adhere.

In the true fibrosarcoma mitoses are rare, for most of these tumors are well differentiated and slowly growing. The finding of giant cells in a true fibrosarcoma is rare and if any significant number of them are seen, a careful study generally identifies the tumor as some other form of sarcoma (liposarcoma, rhabdomyosarcoma, or leiomyosarcoma). Stout recognizes only two forms: the well-differentiated fibrosarcoma with well-developed collagen and reticulin fibers, little pleomorphism or hyperchromatism, and rare mitotic figures; and the undifferentiated fibrosarcoma which has closely packed cells with hyperchromatism, pleomorphism, many mitotic figures, few collagen fibers, and only delicate reticulin fibers.

Recently, several papers describing large series of pulmonary neoplasms have appeared in which passing mention is made of sarcoma. Thornton, Adams, and Bloch³⁴ spoke of one patient with "sarcoma," otherwise unclassified, which was found inoperable at exploration, and was given x-ray therapy but later died. These same authors⁵ subsequently listed 5 unspecified sarcomas discovered among 91 intrathoracic tumors in routine chest fluoroscopy of 15,000 patients in the outpatient department of the University of Chicago Clinics. O'Brien, Tuttle, and Ferkaney,²³ in a report of solitary shadows in the lung, mentioned one patient with "sarcoma" who has since died; no further details are given. Edwards⁸ reported three cases of "sarcoma, all ossifying" in his series of intrathoracic neoplasms, but we can only speculate as to the exact nature of these tumors. The follow-up on these patients revealed that two were operated upon with one postoperative death and one survival for two and one-half years at the time of the report; the third was inoperable but was alive without evidence of disease two and one-half years after a course of x-ray therapy. Harrington¹⁰ mentioned six cases of sarcoma of the lung in which the patients were treated by pneumonectomy, one of whom had survived five years without evidence of recurrence at the time of his report in 1931. The other patients were all dead within two and one-half years of operation.

In their most recent report of patients with pulmonary neoplasms submitted to operation, Ochsner, DeBakey, and associates,²⁴ have had all their microscopic sections reviewed by Dunlap. They had previously reported two cases of fibrosarcoma, but the more recent appraisal placed these two tumors in the class of undifferentiated carcinomas. One tumor, however, still considered a "neurofibrosarcoma," was removed by pneumonectomy with a survival of twelve years. Stout would probably classify this as a fibrosarcoma, but unfortunately for our present purpose the exact site of origin of this tumor is not given. It is the only case in the literature with apparent cure following pneumonectomy, although Neuhof and Aufses²² cited an eleven-year survival

Mallory ¹⁰	13, W, F	2 mo. cough, pleuritic pain, weight loss	Bronchoscopy: Within right main stem bronchus a 1 by 1.5 cm. mass. Post-mortem: Major portion of tumor found lying free in bronchus with evidence of tumor remaining in bronchial wall	Fibrosarcoma growing at moderate rate and invading bronchial wall. No statement about invasion of parenchyma. No silver stain	Died of intrapulmonary hemorrhage during exploratory operation
Pollack et al ¹¹	10, W, M	7 mo. cough, pain, fever, wheezing, weight loss	Bronchoscopy: Left main stem bronchus obstructed 1 in. below carina by soft mass 2 to 3 cm. in diameter. Another similar mass opposite it on bronchial wall. Bronchoscope removal and fulguration on 5 occasions	Fibrosarcoma of bronchus confirmed by Dr. F. W. Stewart of Memorial Hospital N. Y. No silver stain	Wall without bronchoscopic, bronchographic, or x-ray evidence of recurrence 31 mo. later. Well at the end of 6 yr.
Black	46, W, F	9 mo. cough, fever, weight loss, hemoptysis	Operation: Left pneumonectomy. Polypoid 1.5 cm. tumor arising in basal divisions of lower lobe bronchus. Tumor extends through bronchial wall and within 2 mm. of mediastinal pleura; 12 lymph nodes negative. Large abscess in lateral portion of lower lobe with tumor in wall. Post-mortem: Persistent tumor in mediastinal glands, bronchial stump and pericardium	Fibrosarcoma, moderately undifferentiated with pleomorphism and mitotic figures. Wilder stain shows fine reticulin fibers around individual cells	Died of recurrent tumor 6 yr. and 5 mo. after operation

CASE 3 (Lewis¹⁵).—The tumor was removed through the bronchoscope with subsequent radiotherapy. There was persistent tumor four months after treatment, but a recent letter from the author stated that the patient was well with a negative roentgenogram two and one-half years after the institution of treatment.

CASE 4 (Mallory¹⁹).—In only this one case was an attempt at pulmonary resection made, but the patient died of intrabronchial hemorrhage on the operating table.

CASE 5 (Pollack, Cohen, and associates²⁰).—The tumor was removed through the bronchoscope. Thirty-one months after removal and fulguration, which had been performed in 1936, there was no evidence of persistent disease by roentgenogram, bronchogram, or bronchoscopy. A recent personal communication from one of the authors (Cohen) stated that the patient was lost to follow-up study in 1942, at which time he was entirely well. This survival of more than six years is surprising, but owing to the known slow evolution of some fibrosarcomas, even this length of observation may not be adequate to determine the ultimate outcome.

Despite the apparent success of endobronchial forms of treatment in the three cases mentioned, it is not a form of therapy to which we subscribe even though the fibrosarcoma may be a slowly growing tumor. In order to remove all of the neoplasm we feel that the treatment must be by pulmonary resection as in all pulmonary neoplasms. The case reported here illustrates the advantage of pneumonectomy, since the patient had palliation for over six years. In spite of the unfavorable final outcome, it seems justifiable to believe that a cure by radical surgery may be expected in this type of tumor provided it is seen in the early stages of development.

CASE REPORT

M. A. McC. (History No. 93545), a 46-year-old white woman, entered Barnes Hospital on Nov. 12, 1941.* She had a history of several attacks of "flu" nine months previously, characterized by coryza, cough, general malaise, and a low-grade fever. Four months before admission she had another respiratory infection with cough, fever, and small hemoptyses. Treatment with sulfonamides resulted in rapid clearing of the symptoms, but one month later the roentgenograms showed no change in the shadow in the left lower lobe which had been noted previously. Bronchoscopic biopsy was performed, and the husband was informed that the patient had a malignant growth. Repeated sputum examinations and guinea pig inoculations were negative for acid-fast bacilli. Two thoracenteses failed to demonstrate pleural fluid. There was a loss of weight of 10 pounds in four months, but no chest pain at any time.

Past History.—Noncontributory.

Family History.—A maternal aunt died of carcinoma of the breast, and a maternal uncle had carcinoma of the prostate.

Physical Examination.—The patient was a thin, fairly well-developed woman appearing chronically ill but in no acute distress. Vital signs were within normal limits except for pulse of 120. The trachea was in the midline, but the chest seemed to have increased fullness in the left upper region anteriorly with diminution of expansion at the base. There was decreased fremitus and flatness to percussion with absent breath sounds over the entire surface projection of the left lower lobe. No râles were heard, and the right lung field was clear. The remainder of the examination was within normal limits.

Roentgenogram.—The mediastinal structures were in the midline and complete opacity of the lower half of the left lung field as high as the anterior end of the fourth rib was seen. The right lung was clear.

Bronchograms.—Left main stem and left upper lobe bronchi were well filled, but only the apical division of the lower lobe bronchus contained any opaque substance.

*Referred by Dr. John F. Dicks of New Orleans, La.

Fluoroscopy.—There was normal motion of both leaves of the diaphragm.

Laboratory Examination.—Laboratory examination was noncontributory.

Bronchoscopy.—In the left main stem bronchus approximately 1 cm. distal to the upper lobe orifice a polypoid tumor was found completely blocking the lower lobe bronchus. A biopsy showed the obstruction to be due to a very cellular neoplasm.

Operation.—Nov. 17, 1941, under cyclopropane anesthesia, the left sixth rib was resected.* The tumor was located in the lower lobe together with a thick-walled abscess in the lateral peripheral portion. The adhesions between the lower lobe and chest wall were divided without entering the abscess, and the lung was removed, employing the individual ligation technique.

Gross Pathology (Surgical Pathology No. 39049) (Fig. 1).—The surfaces and parenchyma of the left upper lobe were normal. The lingula and lower lobe presented thickened pleura with fibrous adhesions and consolidation. On opening the bronchus a polypoid tumor measuring approximately 1.5 cm. in diameter was found arising from the wall of the lower lobe bronchus occluding it completely. It extended into the subdivisions of the lower lobe bronchus and through the bronchial wall into the surrounding parenchyma. At the inferior portion of the hilum the tumor was growing within 2 mm. of the pleura but had not reached it in any area. The free part of the intrabronchial portion of the tumor reached to within 2.5 cm. of the line of resection.

Distal to the tumor the lower lobe was completely consolidated with many areas having the mottled yellow appearance suggestive of lipoid deposition. At the basal peripheral portion of the lobe there was a ragged abscess cavity measuring 9.0 cm. in diameter and containing necrotic material. The upper lobe was entirely normal. Lymph nodes did not appear to be invaded grossly by the tumor.

Microscopic Pathology.—The predominantly intraluminal character of the tumor and its extension through the bronchial wall toward the pleural surface is illustrated in Fig. 2.† Sections from the wall of the abscess cavity also showed tumor. Fourteen lymph nodes taken from the region of the upper and lower lobe bronchi and two from medial to the main stem bronchus were negative.

The tumor was composed of spindle-shaped cells (Fig. 3) arranged in whorls in some areas. There was moderate variation in the size of the nuclei, some being more rounded than others, and a moderate number of mitotic figures were seen (Fig. 4). Connective tissue stain revealed very little intercellular stroma, and the Wilder stain demonstrated a fine reticulin network wrapped around the individual cells (Fig. 5). The impression was that of a moderately undifferentiated fibrosarcoma arising as a polypoid tumor in the bronchus without demonstrable metastasis or extension beyond the lung, although it had broken through the bronchial wall and invaded the lung parenchyma.

Follow-up.—The postoperative course was marked by a temperature elevation which persisted in the neighborhood of 38° C. for five weeks with occasional spikes to 39.5° and 40° C. Accompanying this there was leucopenia of about 6,000 with a relative lymphocytosis (35 to 50 per cent). Repeated search for malarial parasites because of the fever and enlarged spleen were negative. Pleural fluid cultures were likewise negative, and the temperature elevation was never satisfactorily explained.

After return to her home the patient made a complete recovery except for two brief unexplained febrile episodes occurring two and four years after operation. A letter from her in July, 1947, stated that she was doing housework and feeling well. She spoke of some cough "due partly to allergic irritation of nose and throat" but there was no pain or dyspnea at that time. X-ray examinations of the chest in March and April, 1947, at the Toussaint Infirmary in New Orleans‡ because of returning chest complaints revealed a patchy pneumonic process in the right lung field. However, follow-up films in September, 1947, showed complete clearing of this infiltrate and no other departures from the normal.

*By Dr. Evarts A. Graham.

†These slides, as well as those submitted from the autopsy material, have been reviewed by Dr. Lauren V. Ackerman, Associate Professor of Surgical Pathology, Washington University School of Medicine.

‡Courtesy of Dr. W. R. Arrowsmith, Ochsner Clinic, New Orleans, La.

She remained well until Christmas, 1947, at which time she had an illness diagnosed as "influenza" with afternoon fever as high as 102° F. In March she was hospitalized at the Touro Infirmary at which time the liver was found to be enlarged with a 48 per cent bromsulfalein retention. A needle biopsy of the liver was reported as essentially normal.

Her course was rapidly downhill, and she died April 22, 1948, in the Ochsner Medical Foundation Hospital in New Orleans. Terminally there was paroxysmal auricular tachycardia with a rate of 140-150, and respiratory distress which responded to digitalis, quinidine,



Fig. 1.—Photograph of gross specimen showing polypoid tumor in lower lobe bronchus. Hilus is to the right.



Fig. 2.—Low-power photomicrograph showing origin of tumor from the bronchus in the lower half of the picture (X6).



Fig. 3.—High-power photomicrograph of original tumor showing closely packed fusiform cells with moderate pleomorphism, mitotic figures, and very little intercellular material (X440).

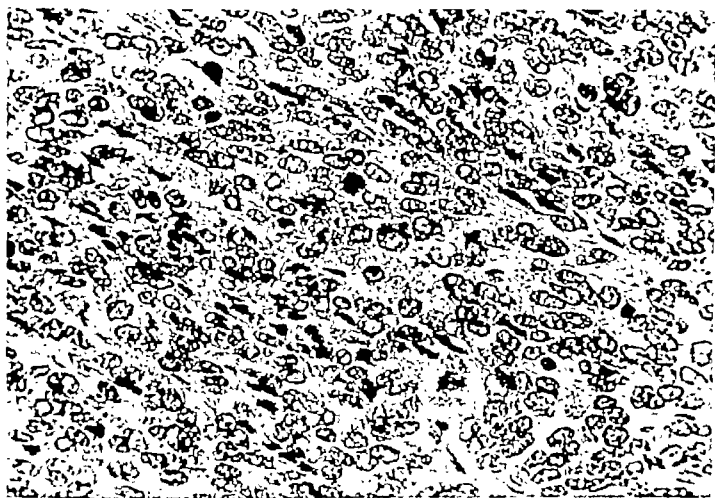


Fig. 4.—Photomicrograph from another area of original tumor which demonstrates mitotic figures and rounded nuclei (X600).

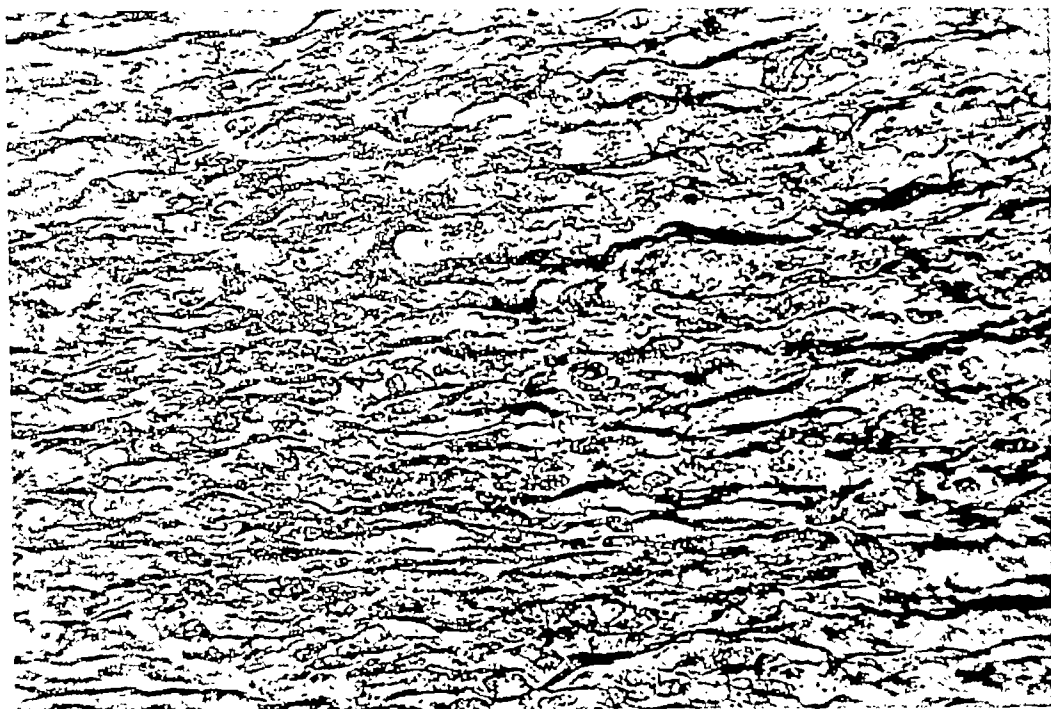


Fig. 5.—Wilder stain reveals fine network of reticulin fibers wrapped around the individual tumor cells ($\times 600$).

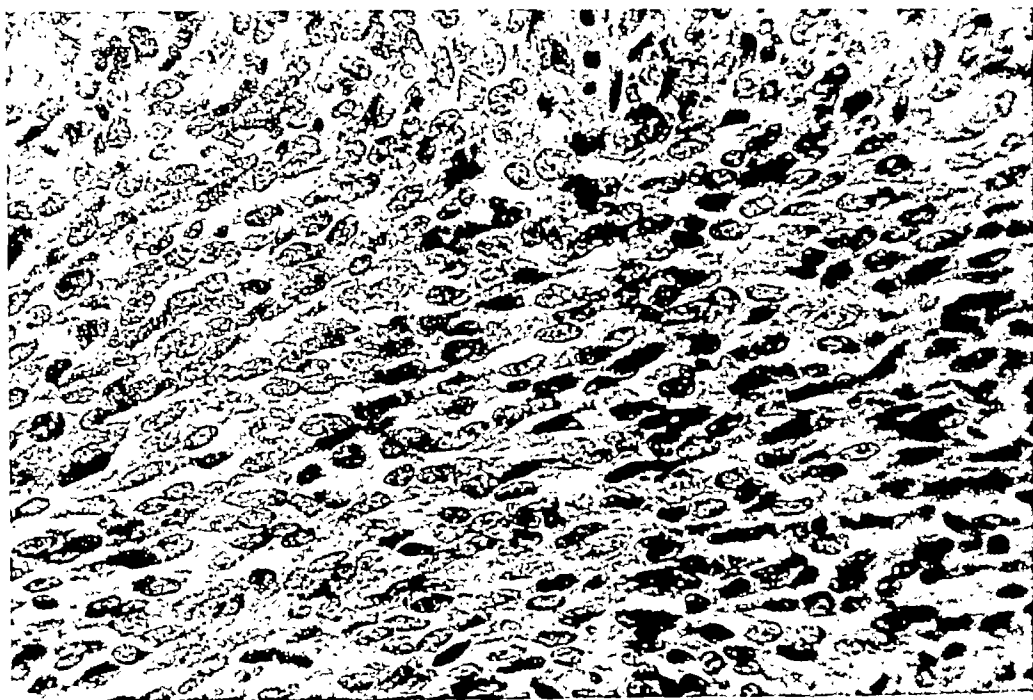


Fig. 6.—Photomicrograph of recurrent tumor at higher power reveals it to be identical with original ($\times 600$).

aminophylline, blood, and oxygen. The electrocardiogram revealed definite evidence of myocardial disease. The liver was enlarged 4 cm. below the costal margin, and there was 2 plus edema of the extremities. Just prior to death there was atelectasis of the right lower lobe, and bronchoscopy revealed external compression of the right main stem bronchus.

She had survived six years and five months after pneumonectomy and seven years and two months after the onset of symptoms, most of this time in good health.

*Post-mortem.**—The mediastinum was shifted markedly to the left and the right pleural cavity contained 1,500 c.c. of serosanguineous fluid. The left pleural cavity was completely obliterated by dense fibrous adhesions. Posterior to the carina there was a large irregular mass which was identified as degenerating tumor in lymph nodes and which extended around the left bronchial stump. It also invaded the hilar area of the right lung, compressing the right main stem bronchus. The tumor mass measured 10 by 8 by 6 cm. and invaded the posterior wall of the left atrium as far as the endocardium, as well as the wall of the esophagus. There was marked distention of the pericardial sac with 750 c.c. of bloody fluid.

Microscopic examination revealed that the tumor was identical with that removed at operation. A representative section is seen in Fig. 6. There were no metastases in the liver, adrenals, pancreas, or elsewhere outside the thorax.

DISCUSSION

That this neoplasm is a rarity in the literature must be obvious from the small number of cases that have been found, but it is equally apparent that this tumor has behaved in a manner similar to fibrosarcomas elsewhere, growing slowly, spreading only by local invasion, and not producing distant metastases. Womack and Graham³² have long held the view that many bronchial and pulmonary tumors arise from the aberrant development of embryologic lung buds and that the resultant neoplasms can therefore consist of derivatives of either entodermal or mesodermal elements in the bronchi or a combination of these. The so-called bronchial adenoma can thus be considered an epithelial variant and the chondroma a mesodermal expression. The neoplasm that is the subject of the present report is believed, from a study of many sections including those from the postmortem, to be a tumor derived from mesodermal tissue only and to be a classical fibrosarcoma. As such it is consistent with this theory of origin of some of the primary bronchial neoplasms, and illustrates a tumor of moderate malignant potentialities.

SUMMARY AND CONCLUSIONS

1. A case of primary fibrosarcoma of the bronchus treated by pneumonectomy is reported. Death occurred from persistent tumor in the thorax six years and five months later.

2. Review of the literature has revealed only five verifiable cases of a similar nature. Although three of these had apparent cure following bronchoscopic removal of the tumors, in one case there was question of persistence at autopsy and in the other two, owing to the known slow evolution of the tumor, the two and one-half and six year follow-up periods, respectively, may not be adequate to determine the ultimate outcome.

3. It is believed that pulmonary resection is the only rational treatment of these neoplasms.

*Description of post-mortem examination and microscopic slides, courtesy of Dr. J. C. Henthorne of the Ochsner Medical Foundation, New Orleans, La.

REFERENCES

1. Adler, I.: Primary Malignant Growths of the Lungs and Bronchi, New York, 1912, Longmans, Green & Co.
2. Ball, H. A.: Primary Pulmonary Sarcoma, *Am. J. Cancer* 15: 2319, 1931.
3. Baum, O. S., Richards, J. H., and Ryan, M. D.: A Case of Atelectasis of the Right Lower and Middle Lobes With Bronchoscopy Demonstrating Spindle Cell Sarcoma of the Right Main Bronchus, *Ann. Int. Med.* 12: 699, 1938.
4. Baum, O. S., Silverman, G., Rosh, R., and Riley, R. L.: A Case of Spindle Cell Sarcoma of the Bronchus; Supplementary Report, *Ann. Int. Med.* 17: 750, 1942.
5. Bloch, R. G., Adams, W. E., Thornton, T. F., and Bryant, J. E.: Difficulties in the Differential Diagnosis of Bronchiogenic Carcinoma, *J. THORACIC SURG.* 14: 83, 1945.
6. Boschowsky, W.: Ueber Primares Lungensarkom, *Frankfurt. Ztschr. f. Path.* 9: 239, 1912.
7. Edwards, A. T.: Intrathoracic New Growths; An Account of Seven Operable Cases, *Brit. J. Surg.* 14: 607, 1927.
8. Edwards, A. T.: Carcinoma of the Bronchus, *Thorax* 1: 1, 1946.
9. Ellis, R. C.: Primary Sarcoma of the Lung With Brain Metastasis, *J. Kansas M. Soc.* 40: 243, 1939.
10. Harrington, S. W.: The Surgical Treatment of Tumors of the Lung and Mediastinum, *Surg., Gynec. & Obst.* 53: 417, 1931.
11. Jackson, C.: Malignant Growths of the Lung, *Arch. Otolaryng.* 12: 747, 1930.
12. Johns, E. P., and Sharpe, W. C.: Primary Sarcoma of the Lung. Case Report, *Am. J. Cancer* 23: 45, 1935.
13. Klemperer, P., and Rabin, C. B.: Primary Neoplasms of the Pleura. A Report of Five Cases, *Arch. Path.* 11: 385, 1931.
14. Lange, J. C.: Four Cases of Malignant Disease of the Lung, *Pennsylvania M. J.* 7: 202, 1903.
15. Lewis, I.: Sarcoma of the Bronchus, *Proc. Roy. Soc. Med.* 40: 119, 1946.
16. Lyssunkin, I. I.: Ueber Primare Pleura—und Lungensarkome, *Frankfurt. Ztschr. f. Path.* 46: 107, 1933.
17. Lyssunkin, I. I.: Zur Frage der Primären Bindegewebegegeschwulste der Lunge, *Beitr. z. path. Anat. u. z. allg. Path.* 94: 491, 1934.
18. Mallory, T. B.: Cabot Case 22441, *New England J. Med.* 215: 837, 1936.
19. Mallory, T. B.: Cabot Case 24202, *New England J. Med.* 218: 843, 1938.
20. Mallory, T. B.: Cabot Case 24472, *New England J. Med.* 219: 854, 1938.
21. Mallory, T. B.: Cabot Case 31271, *New England J. Med.* 233: 18, 1945.
22. Neuhof, H., and Aufses, A. H.: Cancer of the Lung; Interval and Late Results of Operation in Relation to Topography and Gross Pathology, *J. THORACIC SURG.* 17: 297, 1948.
23. O'Brien, E. J., Tuttle, W. M., and Ferkany, J. E.: The Management of Pulmonary "Coin" Lesions, *S. Clin. North America* 28: 1313, 1948.
24. Ochsner, A., DeBakey, M., Dunlap, C. E., and Richman, I.: Primary Pulmonary Malignancy, *J. THORACIC SURG.* 17: 573, 1948.
25. Pilot, I.: Mesenchymatous Tumors of the Lung and Pleura, *Radiology* 14: 391, 1930.
26. Pollack, B. S., Cohen, S., Borroni, M. G., and Gnassi, A. M.: Primary Sarcoma of the Lung. Case Report, *Am. J. Roentgenol.* 41: 909, 1939.
27. Pritchard, J. S.: An Unusual Case of Pulmonary Neoplasm, *Am. J. Roentgenol.* 8: 555, 1921.
28. Rolleston, H. D., and Trevor, R. S.: A Case of Primary Sarcoma of the Lung, *Brit. M. J.* 1: 360, 1903.
29. Scheidegger, S.: Eine Seltene Lungengeschwulst, *Ztschr. f. Krebsforsch.* 35: 172, 1931.
30. Seydel: Ueber Operabilität von Lungen—und pleural—Tumoren, *München. med. Wchnschr.* 57: 452, 1910.
31. Stevens, A. A.: Malignant Disease of the Lung, *Am. J. Med. Sc.* 144: 193, 1912.
32. Stohr, R., and Sachs, W.: Zur Klinik und Pathologie des primären Lungensarkoms. *Deutsche. Ztschr. f. Chir.* 249: 481, 1937.
33. Stout, A. P.: Fibrosarcoma. The Malignant Tumor of Fibroblasts, *Cancer* 1: 30, 1948.
34. Thornton, T. F., Jr., Adams, W. E., and Bloch, R. G.: Solitary Circumscribed Tumors of the Lung, *Surg., Gynec. & Obst.* 78: 364, 1944.
35. Womack, N. A., and Graham, E. A.: Mixed Tumors of the Lung; So-Called Bronchial or Pulmonary Adenoma, *Arch. Path.* 26: 165, 1938.

EXPECTORATION OF A FIBROSARCOMA

PATIENT WELL FOUR YEARS LATER

JOHN J. CURRY, M.D., AND JOB E. FUCHS, M.D.
BOSTON, MASS.

IN RECENT years considerable attention has been given to microscopic examination of expectorated material with a consequent advance in the study of intrapulmonary cancer. In 1945 Samper* reported the case of a patient who coughed up tissue which proved to be a grade 3 squamous-cell epithelioma. Bronchoscopic and lipiodol studies failed to reveal the source of the tumor but four years later, after pneumonectomy, the origin of the cancer was found in the left main bronchus.

The following case is reported because it is unusual and re-emphasizes the importance of microscopic examination of coughed up material.

C. M., a white female, aged 13 years, was referred to the Massachusetts Memorial Hospitals in April, 1944. Except for a mild attack of rheumatic fever she had enjoyed good health until about six weeks before admission to the hospital. At this time she noted a gradual onset of cough associated with intermittent wheezing and productive of slight amounts of yellowish sputum. Three weeks later she experienced a fairly severe and steady nonpleuritic pain in the left side of the chest, close to the sternum. One week before admission, shortly after a bout of strenuous exercise, she coughed up a lump of tissue "about the size of an almond" and then approximately one cupful of bright red blood. Following this episode cough and wheezing disappeared. The tissue was submitted to the Leary laboratory by her family physician and the following was reported: The specimen consisted of a gray-red mass, measuring 1.5 by 1 cm., showing an external smooth gray surface and no demonstrable stalk or base. On section the tissue was uniformly gray-red and resembled fish flesh. Microscopic diagnosis was fibrosarcoma. The patient was then referred to the hospital seven days after the episode of expectoration and was completely asymptomatic.

Physical examination revealed a well-developed and well-nourished young white girl in no apparent distress. Nasopharyngoscopy results were negative. The trachea was in the midline and there were no cervical or axillary nodes. The lung fields were clear and resonant and the remainder of the examination was likewise not remarkable.

Laboratory work showed repeated urinalyses negative. On examination of the blood the level of hemoglobin was 15 Gm. per 100 c.c., leucocytes numbered 9,750 per cubic millimeter, and the differential white cell count was normal. Stools tested gave negative results with gum guaiac. Fluoroscopic examination of the chest showed normal respiratory movement of both diaphragms. X-ray films of the chest taken in inspiration and expiration were also within normal limits. Roentgenograms of skull and sinuses were negative. The hospital course was afebrile and asymptomatic. Bronchoscopic examination was twice performed and revealed a normal carina with a heaping up of tissue just inside the left main stem bronchus on the mesial wall. Only a small biopsy was possible because of the location of the lesion and examination of this tissue revealed granulating fibromuscular tissue.

*From the Evans Memorial, Massachusetts Memorial Hospitals, and the Department of Medicine, Boston University School of Medicine.

Received for publication Aug. 23, 1948.

*Samper, R.: Expectoration of Carcinomatous Tissue Without Localizing Signs in Four Subsequent Years, Pneumonectomy: Report of a Case, Proc. Staff Meet., Mayo Clin. 20: 341, 1945.

†By Dr. John Strieder.

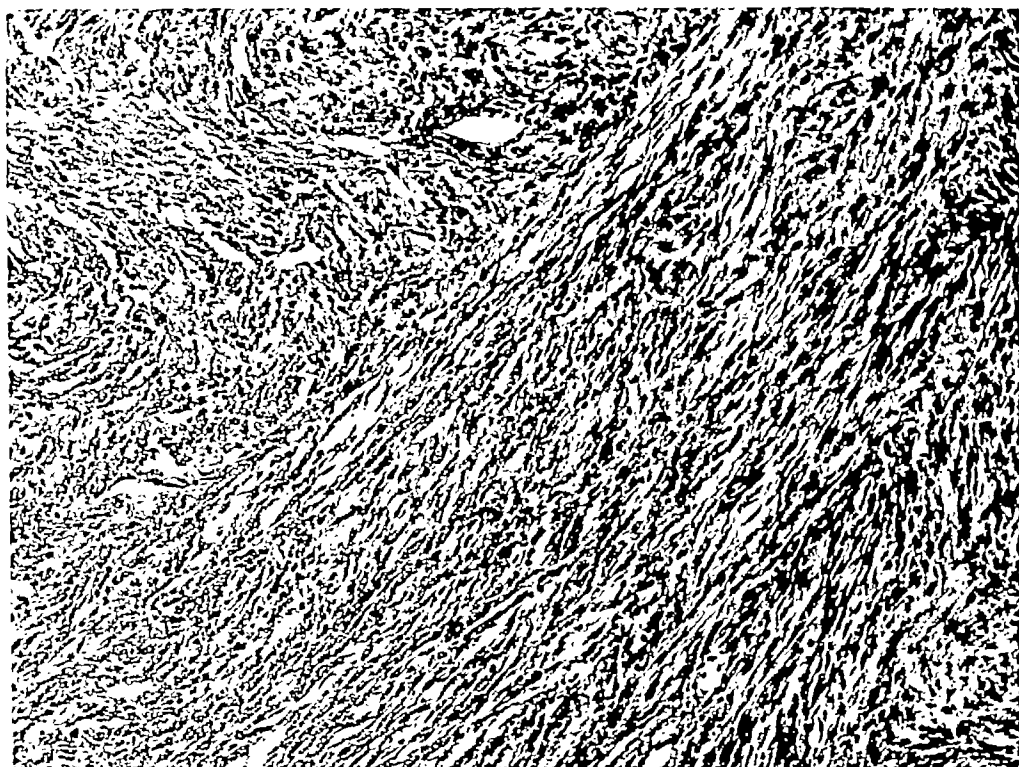


Fig. 1.—Low-power view showing whorled appearance with typical fusiform cells. A tumor giant cell is noted in the center of the field. (Hematoxylin and eosin, $\times 200$.)

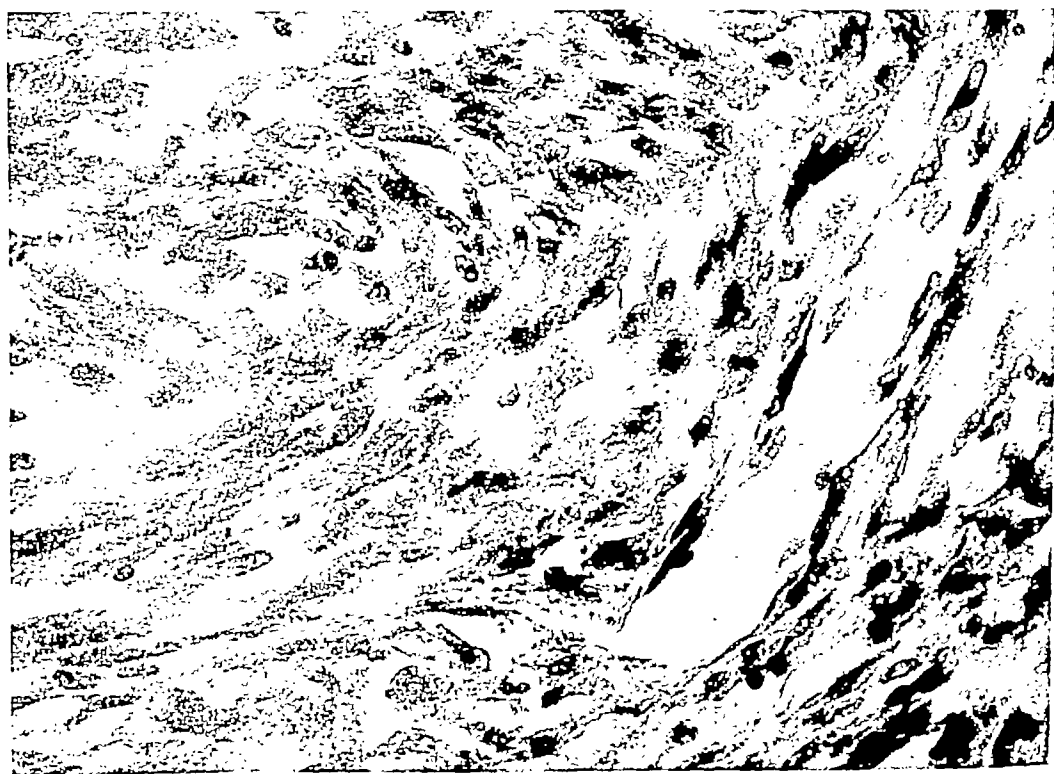


Fig. 2.—Higher magnification displaying two mitoses. The cells are fusiform with variability in nuclear size and staining, and possess a finely granular cytoplasm. A capillary courses through the section. (Hematoxylin and eosin, $\times 800$.)

A review of the original section from the tumor mass showed that it was roughly 2 cm. in diameter and composed of dense interlacing strands of moderately anaplastic fibroblasts. There were numerous mitotic figures, many of which were atypical. A small amount of rather poorly vascular stroma was present. The capsule was sharply limited and along at least three-fourths of its circumference was covered with stratified squamous to transitional epithelium. The diagnosis of rapidly growing fibrosarcoma was confirmed.*

Since the tumor apparently was entirely removed it was decided not to risk pulmonary fibrosis by the administration of x-ray therapy. The patient was therefore discharged from the hospital and one month later readmitted for further bronchoscopic study. There was little change in the appearance of the tracheobronchial tree from the previous examinations.

Follow-up study was carried out on the patient during the ensuing four years and repeated x-ray examinations of the chest were made. The last roentgenogram in July, 1948, was again reported within normal limits and she has continued to be well.

SUMMARY

The case of a young girl who expectorated a rapidly growing fibrosarcoma and made an uneventful recovery is reported. Follow-up of the case over a period of over four years has failed to reveal any recurrence. The importance of a microscopic examination of coughed-up material is re-emphasized.

*Dr. Charles Branch and Frederic Parker of the Mallory Institute of Pathology.

A FOREGUT CYST OF THE MEDIASTINUM

M. A. CASSEL, M.D., R. T. CUNNINGHAM, M.D., AND WILSON WEISEL, M.D.
MILWAUKEE, WIS.

THE finding of alimentary and respiratory tract elements in intrathoracic cysts continues to be an interesting but rare medical and pathologic discovery. A survey of the literature^{2, 10, 14, 16, 18, 21, 34} reveals that probably less than ten cases of cysts containing esophageal, gastric, or enteric and respiratory elements have been reported in the literature.^{6, 24} It is because of the infrequent occurrence as well as the particular pathologic character of the specimen herein described that the following case is being reported.

CASE REPORT.—The patient, a 24-year-old white man, was admitted to this hospital on Aug. 12, 1947. In 1942 a routine x-ray picture of the chest taken for admission to West Point revealed a mediastinal tumor mass projecting to the right. He was sent to an Army General Hospital where deep roentgen therapy was given with no change in the appearance of the lesion. He was discharged from the service in 1943 because of the mediastinal shadow visualized by x-ray. He was followed by repeated x-ray examinations at this and other hospitals with no demonstrable change in the size or character of the shadow.

At the time of admission the patient had no complaints referable to the chest; he did not appear ill and physical examination revealed no abnormal findings. The past history, family history, and occupational history were noncontributory to the diagnosis. The laboratory findings were not significant.

On Aug. 20, 1947, bronchoscopy was performed. This showed a subcarinal widening with elevation of the right bronchus which was thought to be due to a mediastinal tumor.

On Aug. 26, 1947, under endotracheal cyclopropane, ether and oxygen anesthesia, a right thoracotomy was performed* through a posterolateral incision, utilizing an intercostal approach to the pleural space. A reddish-brown cystic mass was found posterior to the lung, and protruding from the right mediastinum just inferior to the azygos vein. The mediastinal pleura overlying the cyst was incised and dissected from the cyst wall, and the azygos vein was divided and ligated for better exposure. The cyst was dissected from its mediastinal bed. There was no apparent stalk nor connection to the trachea, bronchus, or esophagus. However, it was firmly adherent to the vertebral bodies and to dense tissue which extended to the left of the spine. Although no large vessels were encountered in freeing the cyst, there was moderate bleeding after dissecting the cyst from the vertebral bodies. Hemostasis was secured and the mediastinal pleura was closed with interrupted cotton sutures. The lung was re-inflated and the chest wound was closed in layers with interrupted cotton sutures.

The patient's postoperative course was uneventful and he was discharged from the hospital on the eighth postoperative day to continue his university studies. Follow-up examination and roentgenograms have revealed a normal chest.

From the Departments of Surgery and Pathology, Veterans Hospital, Wood, Wis., and the Marquette University School of Medicine, Milwaukee, Wis.

Published with permission of the Chief Medical Director, Department of Medicine and Surgery, Veterans Administration, who assumes no responsibility for the opinions expressed or conclusions drawn by the authors.

Received for publication May 27, 1948.

*By Dr. Forrester Raine.

Pathologic Examination.—

Gross description: The specimen (Fig. 1) consisted of a cyst measuring 5.5 by 3.5 cm. after formalin fixation. The cyst wall averaged 2.0 to 4.0 cm. in thickness. The inner surface was corrugated and grayish-yellow and coated by a thick viscid mucous fluid. Section through the wall showed a mucosal, a muscular, and an outer layer.

Microscopic description: The wall of the cyst contained the inner two layers of intestinal tract wall. There was a distinct mucosa, with submucosa, an inner and outer muscular coat, and an adventitial fibrous layer. The primary sections which were cut revealed a gastric type of epithelium lining the mucosa. However, after multiple sections were cut from different areas of the cyst wall, three types of epithelium could be identified. In some areas esophageal mucosa comprised of noncornifying squamous-cell epithelium was seen which merged into a respiratory epithelium consisting of ciliated, goblet cells. In other areas there was a poorly defined mucosa which was taller than the other two. The cells were poorly outlined, but seemed to have the appearance of stratified mucous glands such as might be seen in the stomach (Fig. 2, *A*, and *B*). In the submucosa there were collec-

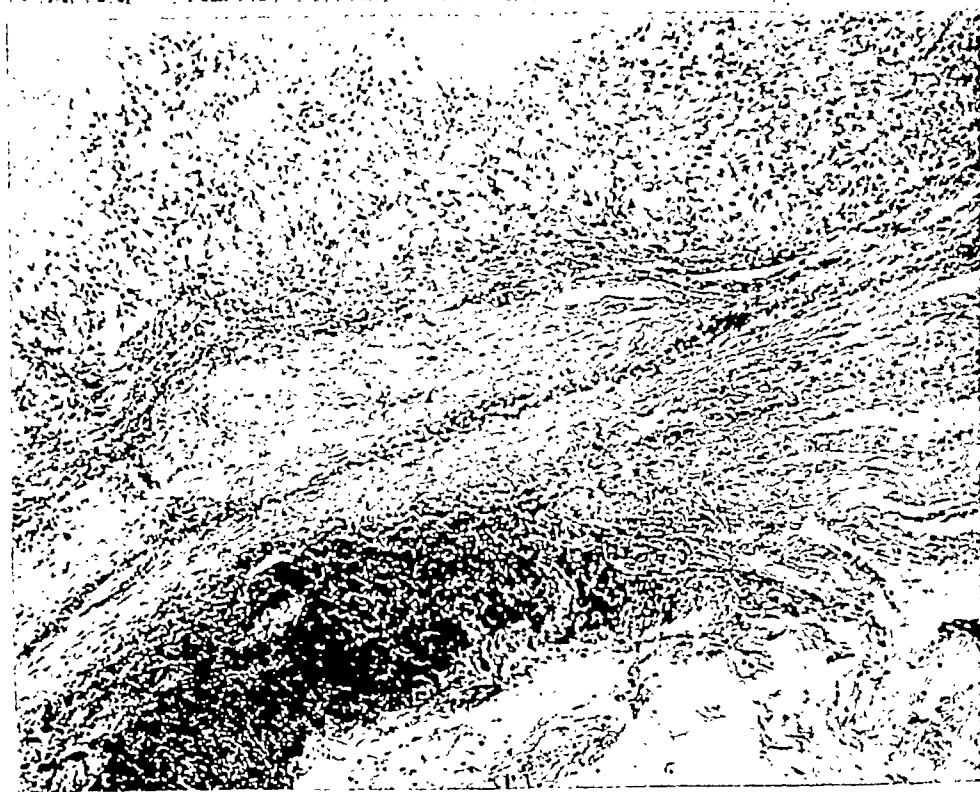


Fig. 1.—Gross specimen of foregut cyst showing mucosal lining.

tions of lymphocytes in both follicular and diffuse formation. The musculature showed distinct circular and longitudinal muscle bundles. The outer layer contained fibrous tissue and blood vessels.

The diagnosis was foregut cyst of the mediastinum.

A.



B.

Fig. 2.—A, Photomicrograph (low power) showing transition between esophageal and bronchial epithelium; smooth muscle is seen below the mucosa. B, Epithelial lining resembling degenerated gastric mucosa.

DISCUSSION

The origin of these cysts has long been a subject of speculation by numerous investigators. Their occurrence primarily in children and the resemblance of the epithelial elements to embryonic prototypes have caused most workers interested in the subject to turn to embryologic maldevelopment as a probable explanation of their genesis. There have been many theories advanced attempting to explain these anomalies. These concepts have been:

1. Origin from embryonic diverticula of the alimentary system or foregut. This hypothesis is considered most likely by the majority of authors.
2. Origin from vestigial remnants of the omphalomesenteric system or vitelline duct.
3. Failure of coalescence of the vacuoles in the transverse axis when the alimentary tube is in the solid stage. Thus, a cystic structure is left contiguous to some portion of the alimentary tract.
4. Sequestration of embryonic multipotential entodermal cells from the lung bud area of the primitive alimentary anlage, with later growth and development.
5. Imbalance of organizational substances in embryonic life.
6. Abnormal or isolating twinning theory of origin similar to dermoids and teratomas.

It is clear that no single theory of origin has been found completely acceptable for mediastinal cysts,²⁷ and various pathologic processes, including metaplasia and neoplasia, have been invoked to explain the two different histologic findings that have been described.

The histologic pattern of the cyst in our case presents an epithelium from both the alimentary and bronchial systems. The primitive foregut has potentialities of developing into both respiratory and enteric epithelium. During the fourth week of development of the embryo, lung buds develop as an evagination from the esophagus. At this embryonic stage the pinching off of an outbud or diverticulum of foregut could be carried along by the downward growing lung bud and lodge in the mediastinum or it may remain attached to its esophageal origin. We are, therefore, in accord with those writers who ascribe these anomalies to diverticula of the embryonic foregut.

The histologic character and differentiation of the various mediastinal cysts has been adequately described by a number of authors.^{2, 3, 13, 16, 19} In describing the bronchiogenic cysts, most writers have considered not only the characteristic epithelium, but have stressed the finding of cartilage in the wall of the cyst. The comparative incidence of bronchiogenic cysts was illustrated by the series of ninety-four cases of benign mediastinal tumors reported by Blades,² in which twenty-three of the tumors were bronchiogenic cysts and twenty were teratoid types. It is possible that a definite classification of these

cysts may be impossible, due to complete destruction or dissolution of the lining, as was found in the case reported by Donald.⁸ In reviewing, we attempted to evaluate the effect of the roentgen therapy which the patient has received upon the pathologic findings, but we are unable to demonstrate any definitive features that we could ascribe to the radiation.

In the specimen described, the most interesting pathologic feature was the varied and transitional character of the epithelium, which was seen in the mucosa. A combination of the respiratory or ciliated columnar epithelium and the alimentary or stratified squamous and mucous gland type of mucosa has been reported previously,^{24, 29, 32} but it has not been frequently described in the literature. It would, perhaps, be more commonly seen if many sections were examined from the so-called bronchial or gastric cysts, or esophageal duplications.

The asymptomatic nature of the case reported has been a finding rather commonly reported by others and indicates nonfunction of the mucosal elements.^{2, 14, 28, 31, 32} The fact that these cysts, however, may frequently cause symptoms and lead to a variety of complications has been stressed by a large number of workers. The common symptoms that have been described in association with mediastinal cysts of this type have been cough, dyspnea, cyanosis, hemoptysis, pleuritic pains, recurrent bouts of pneumonia, dysphagia, fever, and malaise. Most of the complications arising from the cysts that have been described were found to occur in children. These have consisted of rupture into a bronchus, pleural space, or the trachea, often due to perforation of a peptic ulcer arising from gastric mucosa,^{1, 6, 7, 26} erosion of the vertebra or ribs or erosion of the esophagus, and, secondarily, they have been the cause of atelectasis, pneumonitis, empyema, and bronchiectasis.⁷ Many other anomalies that were usually developmental in nature have been described in association with these lesions and the cysts have also been described as extending down to and below the diaphragm into the stomach.^{7, 32}

The treatment of choice of the posterior mediastinal cyst is undoubtedly extirpation in a one-stage procedure if this is possible. Heuer and Andrus¹⁴ have stressed the danger of neglecting these lesions, and the importance of operation both from a diagnostic and therapeutic standpoint. These authors have further emphasized the hazard and futility of subjecting their patients to preoperative irradiation because of the lack of therapeutic response and the fibrous reaction resulting from this procedure. The risk of the operation has lessened in the past two decades to a point that the danger of thoracotomy is not as great as the risk created by the presence of the tumor.

The procedure used in this patient illustrates the type of operative approach we prefer in lesions of the posterior mediastinum. We have felt that the posterolateral incision with or without the removal of a rib has given adequate exposure to this region and the postoperative recovery has been excellent in these patients.

SUMMARY

1. A foregut type of posterior mediastinal cyst occurring in a 24-year-old white man is presented.

2. The pathologic examination of the cyst revealed a lining epithelium similar to that found in the respiratory tract, esophagus, stomach, and intestines.

3. The cyst was removed in a one-stage operation through a posterolateral approach with complete recovery.

The authors are indebted to Mr. Leo Massopust, Marquette University School of Medicine for the Photomicrographs and to Dr. Joseph M. Lubitz, Pathologist, Veterans Hospital, Wood, Wis., and Marquette University School of Medicine, for his valuable assistance in the preparation of this report.

REFERENCES

1. Black, R. A., and Benjamin, E. L.: Enterogenous Abnormalities; Cysts and Diverticula, *Am. J. Dis. Child.* 51: 1126-1137, 1936.
2. Blades, Brian: Mediastinal Tumors, *Ann. Surg.* 123: 749-765, 1946.
3. Brown, R. K., and Robbins, L. L.: The Diagnosis and Treatment of Bronchogenic Cysts of the Mediastinum and Lung, *J. THORACIC SURG.* 15: 84-105, 1944.
4. Carlson, H. A.: Congenital Cysts of the Mediastinum; Report of Three Cases, Including a Gastric Cyst, *J. THORACIC SURG.* 12: 376-393, 1943.
5. Davidson, L. R., and Brown, Lowell: Gastrogenous Mediastinal Cyst, *J. THORACIC SURG.* 16: 458-467, 1947.
6. Davis, E. W., and Salkin, David: Intrathoracic Gastric Cysts, *J. A. M. A.* 135: 218-221, 1947.
7. Dickson, J. A., Clagett, O. T., and McDonald, J. B.: Intrathoracic Gastric Cyst, *J. THORACIC SURG.* 15: 318-323, 1946.
8. Donald, Charles J., Jr.: Mediastinal Cysts, *South. Surgeon* 13: 148-151, 1947.
9. Entz, B., and Orosz, D.: Ueber die Intrathorakalen Zystenbildungen, *Frankfurt. Ztschr. f. Path.* 40: 229-246, 1930.
10. Fischer, Walther: Ueber eine Cyste in der rechten Pleurahohle, *Virchows Arch. f. path. Anat.* 275: 711-716, 1930.
11. Fitz, Reginald H.: Persistent Omphalomesenteric Remains; Their Importance in the Causation of Intestinal Duplication, Cyst Formation, and Obstruction, *Am. J. Med. Sc.* 88: 30-37, 1884.
12. Freeland, S. O., and Gebauer, P. W.: Disease of Aberrant Intrathoracic Lung Tissue, *J. THORACIC SURG.* 8: 581-597, 1939.
13. Greenfield, L., Sternberg, L., and Touroff, A. S. W.: Spring Water Cyst of the Mediastinum, *J. THORACIC SURG.* 12: 495-502, 1943.
14. Heuer, G. J., and Andrus, W. DeW.: The Surgery of Mediastinal Tumors, *Am. J. Surg.* 50: 143-224, 1940.
15. Keith, A.: *Human Embryology and Morphology*, ed. 5, Baltimore, 1933, William Wood & Company, p. 558.
16. Ladd, W. E., and Gross, R. E.: Surgical Treatment of Duplications of the Alimentary Tract, Enterogenous Cysts, Enteric Cysts or Ileum Duplex, *Surg., Gynec. & Obst.* 70: 295-307, 1940.
17. Ladd, W. E., and Scott, H. W.: Esophageal Duplications or Mediastinal Cysts of Enteric Origin, *Surgery* 16: 815-835, 1944.
18. Laipply, T. C.: Cysts and Cystic Tumors of the Mediastinum, *Arch. Path.* 39: 153-161, 1945.
19. Lambert, A. V.: Etiology of Thin-Walled Thoracic Cysts, *J. THORACIC SURG.* 10: 1-7, 1940.
20. Lewis, F. T., and Thyng, F. W.: The Regular Occurrence of Intestinal Diverticula in Embryos of the Pig, Rabbit and Man, *Am. J. Anat.* 7: 505-510, 1908.
21. Lindquist, Nils, and Wulff, H. B.: Mediastinal Enterocystoma, *J. THORACIC SURG.* 16: 468-476, 1947.
22. Mixer, C. G., and Cliffford, S. H.: Congenital Mediastinal Cysts of Gastrogenic and Bronchogenic Origin, *Ann. Surg.* 90: 714-729, 1929.

23. Nicholls, M. F.: Intrathoracic Cyst of Intestinal Structure, *Brit. J. Surg.* 28: 137-143, 1940-41.
24. Olenik, J. L., and Tandatnik, J. W.: Congenital Mediastinal Cyst of Foregut Origin, *Am. J. Dis. Child.* 71: 460-476, 1946.
25. Olken, H. G.: Congenital Gastroenteric Cyst of the Mediastinum, *Am. J. Path.* 20: 997-1009, 1944.
26. Poncher, H. G., and Milles, G.: Cysts and Diverticula of Intestinal Origin, *Am. J. Dis. Child.* 45: 1064-1078, 1933.
27. Rusby, N. L.: Dermoid Cysts and Teratomata of the Mediastinum; A Review, *J. THORACIC SURG.* 13: 169-222, 1944.
28. Schwarz, H., and Williams, C. S.: Thoracic Gastric Cysts, *J. THORACIC SURG.* 12: 117-130, 1942.
29. Smith, R. E.: A Case of Mediastinal Dermoid Cyst in an Infant, *Guy's Hosp. Rep.* 80: 466-469, 1930.
30. Staehlin-Burckhardt, A.: Ueber eine mit Magenschleimhaut versehene Cyste des Osophagus, *Arch. f. Verdauungskr.* 15: 584-603, 1909.
31. Steele, J. D., and Schmitz, J.: Mediastinal Cyst of Gastric Origin, *J. THORACIC SURG.* 14: 403-406, 1945.
32. Valle, A. R., and White, M. L.: Thoracic Gastric Cyst, *Ann. Surg.* 123: 377-383, 1946.
33. Ward, I. M., and Krah, J. B.: Enterogenous Pulmonary Cyst, *Am. J. Dis. Child.* 63: 924-933, 1942.
34. Wyllie, W. G., and Pilcher, R. S.: Intrathoracic Cysts of Intestinal and Bronchial Structure, *Arch. Dis. Childhood* 18: 34-40, 1943.

THE TREATMENT OF SIMPLE SPONTANEOUS PNEUMOTHORAX

P. C. CURTI, PAVIA, ITALY, AND THUE POULSEN, COPENHAGEN, DENMARK

SIMPLE spontaneous pneumothorax is a well-defined clinical syndrome exhibiting the following characteristics: The pneumothorax occurs spontaneously (in contradistinction to artificial or traumatic pneumothorax) and not as a complication of any other marked diseases (inflammatory or tumorous) of the lung, trachea, esophagus, or the thoracic wall, and, finally, the disease is not accompanied by secondary infection.

The etiology may vary, a point which it is of the utmost importance to bear in mind when working out the method of treatment. The commonest cause seems to be spontaneous rupture of subpleural valvular cysts. Evidence to this effect was first presented in this country by Kjaergaard (1932) on the basis of nine autopsy findings (six cases reported in the literature, three cases personally observed). The author describes two different types. In one type the valve is formed by emphysematous tissue, in the other by scar tissue. An additional two cases are reported in a later publication. In either case autopsy revealed multiple congenital cysts. In one case the cysts communicated with the pleural cavity through a small aperture. The observations of Kjaergaard have been confirmed by numerous other authors (Gordon; Grapengiesser; Sycamore; Castex and Mazzei; Mourier; Gullbring and others). On the basis of studies of familial cases the possibility was suggested that congenital defects in the pulmonary tissue may be a contributing cause of the formation of subpleural vesicles with ensuing pneumothorax (Götzsche; Orrego Puelme and Richard).

Cutler reports cases of spontaneous pneumothorax which occurred in the course of artificial pneumothorax therapy and were cured after cauterization of adhesions. He is, therefore, of the opinion that the causative factor may be small fissures in the visceral pleura brought about by the traction of adhesions. As regards our Case 2 this explanation seems obvious.

Based on anatomical, clinical, and experimental studies Macklin advanced the theory that pneumothorax may develop in consequence of a small fissure in the mediastinal pleura which in the presence of interstitial emphysema may be exposed to overdistention and may burst.

The mechanical factor responsible for the rupture of vesicles is assumed to be the increased pressure which occurs under forced expiration in the presence of a valvular system (Macklin).

The symptoms and the objective manifestations of the disease are well-known and we shall confine ourselves to re-emphasizing that to some extent the symptoms are proportional to the suddenness at which the air enters the pleural cavity and the actual amount of air involved. In some cases the symptoms are hardly evident, or absent. Roentgenographic examination, however, makes it possible to disclose the presence of even quite small quantities of air, which are

distinctly observable under forced expiration. In some cases roentgenography has furthermore made it possible to detect subpleural vesicles or cysts (Kjaergaard; Gordon; Sycamore; Castex and Mazzei). By this means cysts were revealed in two of the cases studied by us (Cases 5 and 8).

Roentgenographic examination is usually an insufficient means of detecting minor cysts which do not show up owing to their small size, their location or the thinness of the cyst wall. In the more recent literature a few authors have mentioned thoracoscopy as a valuable aid in detecting the presence of vesicles (Sycamore; Castex and Mazzei; Mourier; Gullbring; Begtrup-Hansen; Brock).

Routine thoracoscopic examination was carried out in all our cases except two in which roentgenography revealed the presence of cysts.

In case of complete pneumothorax where the location of the process is usually unknown it is advisable to insert the thoracoscope in the fifth or sixth intercostal space in the posterior axillary line or in the midaxillary, from where one will obtain an excellent view of the surface of the lung. The picture presenting itself is most characteristic and cannot possibly be mistaken for any other lesion of this area, as it will reveal a smaller or larger number of thin-walled transparent emphysematous blebs of varying size localized within a more or less extensive area. Sometimes a fistula is seen (Case 6), but it will generally be closed owing to the collapse of the lung. One cannot in all instances expect to obtain a complete view of the extent of the process, as it is not always possible to look into the fissures and the mediastinal surface. Thus, in Cases 4 and 7 the process appeared to be far more extensive than revealed by thoracoscopy. The fact that it is impossible to view the entire pleura and thus ascertain the extent of the process will undoubtedly necessitate exploratory thoracotomy in some cases (frequently recurring pneumothorax, chronic pneumothorax) even though thoracoscopic examination is negative.

Considering the results obtained in cases reported in the literature and in our own material and in view of the minimum risk involved by thoracoscopy, the institution of routine thoracoscopic examination is recommended in cases of spontaneous pneumothorax.

The prognosis of spontaneous pneumothorax is generally regarded as favorable with a tendency to spontaneous resorption of the air and re-expansion of the lung within a few weeks, but the increased number of cases observed and the prolonged period of observation have afforded knowledge of the serious complications which may occur in the course of the disease, and it is, therefore, no longer justifiable to regard the disease as absolutely benign.

The most frequent complication is the recurrence of attacks. Cohen and Kinsman observed recurrence of symptoms in 10 per cent, Wilson in 20 per cent, Kjaergaard in 15 per cent, Ornstein and Lercher in 30 per cent, Wood in 45 per cent and Schneider and Reissman in 20 per cent of the cases studied by them. In our material recurrence was observed in five cases of eight. The number of recurrent attacks is greatly varying, from one or two (as seen in the majority of cases) to numerous. Thus, Locke reports a case of eighteen recurrent attacks. Also the intervals vary greatly. It is obvious that the patient may

be troubled by recurrent attacks for a number of years. In recurrent attacks the symptoms are frequently mild or indefinable and are sometimes only manifested by slight functional dyspnea which makes it difficult to establish a correct diagnosis (Schneider and Reissman). It is, therefore, likely that the incidence is higher than generally assumed.

In other cases the spontaneous pneumothorax becomes chronic, for example, when traction of adhesions prevents healing of the fissure (Kjaergaard; Hoyer and Clagett; Mainini and Alvarez; Babington; Schoner and Erlich). In the presence of fairly large accumulations of air this may give rise to a permanent feeling of oppression of the chest and dyspnea. The patients are frequently weak and miserable and are progressively losing weight which makes them look like hunger patients (Brock).

The presence of a valvular system always involves the risk of the patient's developing tension pneumothorax, always a serious condition which, if untreated, may result in death. Kjaergaard observed this complaint in 10 per cent of his cases, Movitt in 16 per cent. Cases of bilateral tension pneumothorax are reported by Wood and by Emerson and Beeler.

Finally, there are occasional occurrences of pneumothorax complicated by hemothorax; Kjaergaard reports two cases out of fifty-one.

The fact that Kjaergaard, in reviewing the literature, found that death was reported in six cases out of 200, serves to prove that the mortality rate is not quite negligible. In his own material there are no cases in which death may be attributed entirely to the existing pneumothorax, but in two cases the pneumothorax was complicated by other lesions of the lungs and may safely be regarded as a contributing cause. In one case the patient developed pneumonia of the collapsed lung and, subsequently, pyopneumothorax; in the other case a left-sided tuberculosis was treated with artificial pneumothorax when the occurrence of a right-sided spontaneous pneumothorax resulted in death.

TREATMENT

As the ordinary conservative therapy consisting of complete rest in bed for some weeks and, in cases of mediastinal displacement and tension pneumothorax, exsufflation, often proves to be insufficient even as the initiation of more radical treatment, we shall give an outline of the method by which these patients should be treated in the acute stage of illness.

When the diagnosis has been established intrathoracic pressure should be measured. If the pressure is elevated, aspiration should be resorted to until the manometer registers zero. Aspiration is likewise indicated in cases where the lung is totally collapsed, even though there is no elevation of the pressure. The patients should now be kept under constant observation and it may be necessary to carry out repeated aspirations or, even better, to insert a drainage tube (Bülau drainage) ending under water or connected with a permanent suction device. The drainage tube should preferably be inserted in the second or third intercostal space anteriorly. The application of drainage averts the danger of disastrous conditions occurring and further treatment may be considered.

Knowledge of the above-mentioned quite frequent complications made some authors advocate a more active therapy. They aimed at complete agglutination of the two pleural membranes which was attained through the production of chemical pleuritis by means of irritant substances. Hypertonic solution of glucose, blood and iodine talcum were applied for this purpose (Hemmel; Bethune; Kulke; Singer and associates; Watson and Robertson). According to Movitt and associates hypertonic glucose is of doubtful value as, in some cases, it gives rise to great exudate but no adhesions. Injection with blood may lead to the formation of fibrin clots preventing re-expansion of the lung. Iodine talcum and similar kinds of powder produce severe pleuritis with marked thickening of the pleura which becomes liable to contract, another factor preventing re-expansion of the lung. In patients treated by this method autopsy findings revealed thickenings of 1 cm. (reported by Ross and Fullerton).

The application of silver nitrate yielded more favorable results. This method was introduced by Brock in 1932. While the patient is lying on the back about 0.5 to 1 c.c. of silver nitrate in 10 per cent solution is injected into the third intercostal space and at the same time exsufflation is carried out. During the first two or three days there is an accumulation of fluid which is aspirated. Fever and pain persist for four or five days. The pleura is said to become dry within ten to fourteen days. By this method Brock treated eighteen patients, twelve with chronic pneumothorax, six with recurring pneumothorax. Complete recovery is reported in all cases. Frequently injections had to be administered two or three times.

THE AUTHORS' MATERIAL

For the past two years we have treated eight cases of simple spontaneous pneumothorax in the Department of Thoracic Surgery of the Rigshospital. In seven of these cases subpleural vesicles or pulmonary cysts were detected, and in one case a small aperture of a fistula was observed immediately beneath an adhesion. In two cases only did roentgenographic examination reveal the presence of cysts, in the remaining cases it was necessary to carry out thoracoscopy in order to be able to establish a correct diagnosis. Of these eight patients four were women aged 20, 21, 26, and 30 years, respectively, and four were men aged 24, 29, 42 and 55 years, respectively. In all these cases the cysts were located in the upper lobe particularly in the lower portion of the anterior margin. There were five instances of left-sided involvement, three instances of right-sided involvement. Three cases were treated with silver nitrate and in five cases pulmonary resection was carried out.

TREATMENT WITH INJECTION OF SILVER NITRATE (THREE CASES)

In three cases we used the method of Brock, slightly modified. When the lesion had been localized by means of thoracoscopy we inserted a long cannula into the thoracoscope and injected 0.5 to 1 c.c. of silver nitrate in 10 per cent solution into the surface of the lung corresponding to the location of the lesion. This part of the procedure is most painful and is carried out under general anesthesia.

CASE 1.—E. H., a man, aged 42 years, had previously been in good health.

On June 9, 1946, he suddenly experienced heavy pain in the left side of the chest with great dyspnea and had to go to bed. The roentgenographic examination which was carried out a couple of days later disclosed complete collapse of the entire left lung. Four months later complete re-expansion of the lung was observed. On Jan. 11, 1947, the patient experienced a similar attack while starting a motorcar, also this time the attack was accompanied by great dyspnea. On January 13, roentgenographic examination revealed a great left-sided pneumothorax with displacement of the heart and mediastinum toward the right. On January 14, thoracoscopy was carried out. Small emphysematous blebs were observed in the anteroinferior portion of the upper lobe, near the fissure. About 1 c.c. of silver nitrate in 10 per cent solution was sprinkled onto this location. On follow-up examination in July, 1947, the patient had recovered completely. Roentgenography showed complete re-expansion of the lung and there was no evidence of exudate or thickening of the pleura.

CASE 2.—J. J., a man, aged 24 years, was admitted to the hospital on April 4 and discharged on April 25, 1947.

He had been hospitalized elsewhere twice in 1944 and once in 1945 for spontaneous pneumothorax. He received conservative treatment. The onset was sudden and could not particularly be ascribed to exertion. The attacks were accompanied by violent pain in the right side of the chest, radiating into the arm, and the patient was suffering from dyspnea and a dry cough. No previous manifestations of tuberculosis had been noted.

On April 3, 1947, the patient experienced a recurrent attack in consequence of which he was admitted to this hospital. Roentgenographic examination revealed a large right-sided pneumothorax with no displacement or exudate. On April 8, thoracoscopy was carried out. No cysts or blebs were seen, but on the other hand a wide adhesion extending from the apex to the top of the cupola was observed. Beneath this adhesion there was a minor cavitation containing a small amount of serosanguineous fluid. On the assumption that this had been the site of a small ruptured bleb about 1 c.c. of silver nitrate in 10 per cent solution was injected onto this location.

A roentgenographic check on April 15 revealed almost complete re-expansion of the lung.

On May 5, 1948, however, a large pneumothorax was once more observed, wherefore exsufflation was carried out.

On follow-up examination on May 28 the patient was feeling well and complete re-expansion of the lung was observed.

CASE 3 (Figs. 1 and 2).—E. L., a man, aged 55 years, was admitted to hospital on June 10 and discharged on July 22, 1947.

During a coughing fit in November, 1946, the patient suddenly experienced violent pain in the right side of the chest, accompanied by great dyspnea. The attending physician ascertained the presence of a right-sided pneumothorax; roentgenographic examination was not done. In the following days repeated aspirations were carried out and after one month in bed the patient was feeling quite well. In May, 1947, he experienced another attack of exactly the same nature and was admitted to the hospital. Roentgenographic examination revealed a total right-sided pneumothorax with displacement of the mediastinum.

On June 10 the patient was admitted to the Rigshospital. He was lean, weak, and anorectic. Thoracoscopic examination the following day revealed collapse but no atelectasis of the lower and middle lobes. Numerous wide adhesions were seen over the upper lobe which obscured the view considerably, but no cysts or fistulas were seen. About 1 c.c. of silver nitrate in 10 per cent solution was injected, a drainage tube was inserted into the cavity and permanent suction applied.

The following days the temperature ranged between 38.5 and 40° C. Roentgenographic examination on June 16 showed that a large pneumothorax persisted in the upper part of the lung. Thoracoscopy was therefore repeated on June 17, this time high in the axillary region. The lower part of the lung had become re-expanded, but in the upper part a cavity

with numerous adhesions persisted. A second injection of about 1 c.c. of silver nitrate in 10 per cent solution was administered and another drainage tube was inserted into the top of the axillary.

As there was a persistent rise of temperature, penicillin and sulphathiazole were administered as from June 26, whereupon the temperature dropped to normal. A roentgenographic check on August 8 revealed a fairly large exudate but no pneumothorax.



Fig. 1.

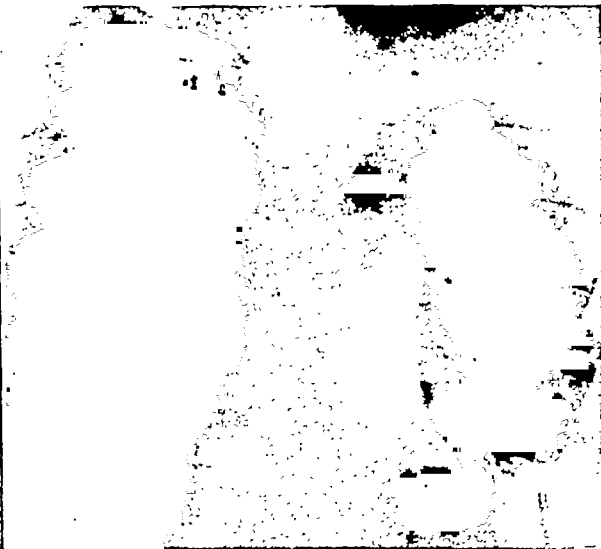


Fig. 2.

Figs. 1 and 2 (Case 3).—Roentgenograms immediately before and eight months after silver nitrate therapy. Fig. 1 reveals a pneumothorax in the upper part of the right lung with displacement of the trachea toward the left. Fig. 2. The roentgenogram shows an organized exudate over the apex of the lung with displacement of the trachea towards the right.

Silver nitrate therapy yielded most satisfactory results in two of the three cases so treated, whereas in the third case there was an organized exudate with displacement of the trachea. There is frequently a violent response to the injection in the form of fever which may persist for several weeks. The treatment is always painful and in our opinion it is more disagreeable than the operative method.

Even though results are satisfactory in most cases this is, however, only a symptomatic method of treatment which does not remove the causative factor in the lung. The drawbacks of the methods of producing chemical pleuritis may be summarized as follows: (1) incomplete re-expansion, (2) frequently severe pleural changes and, finally, (3) a combination of the two.

The latter drawback is, we presume, not quite unusual and may lead to development of chronic pneumothorax which will be resistant to any form of treatment by more conservative methods. A case of this nature will be described in the following. Since the patient received primary treatment in another hospital the case is not included in our material.

CASE HISTORY.—A. A., a girl aged 12 years, was admitted to the hospital on April 29 and discharged on May 17, 1947.

On May 18, 1945, while on a bicycle trip the patient suddenly became affected with great dyspnea accompanied by indefinable pain in the chest. The attack subsided spontaneously in the course of a few hours. A fortnight later the patient experienced a similar

attack because of which she was admitted to a hospital elsewhere. A large left-sided pneumothorax was observed with displacement of the heart and mediastinum. Numerous aspirations proved ineffectual.

On Aug. 20, 1945, thoracoscopy revealed collapse of the lung but no atelectasis. The upper lobe and the upper part of the lower lobe communicated medially with the thoracic wall through numerous wide adhesions. No fistula aperture was seen, but at the base of one adhesion there was a small emphysematous bulla. Following cauterization of the adhesion the lung appeared to be detached.

Röntgenographic examination on November 6 revealed complete re-expansion of the lung.

The patient was now doing well until May 28, 1946, when the presence of a large pneumothorax was once more detected. 2.5 c.c. of silver nitrate in 10 per cent solution was injected under ethyl chloride anesthesia. In the following days repeated aspirations were carried out. On the twelfth day 150 c.c. of brownish serous liquid was aspirated. Despite repeated subsequent aspirations a pneumothorax persisted over the upper part of the lung.

On April 29 the patient was admitted to the Department of Thoracic Surgery of the Rigshospital, where it was assumed that pleural changes were responsible for the failure of the lung to re-expand.

On May 1 thoracotomy with decortication of the lung was carried out. A fairly large pneumothorax was pushing the upper lobe and the upper part of the lower lobe downward. Either lobe was covered by pleural thickenings preventing re-expansion of the lung. No fistulus or vesicles were seen. The lung was dissected free all the way round and excision of the inferior ligamentum pulmonale was performed. Subsequently the lung became more mobile but failed to become completely re-expanded; the thickened pleura over the upper and lower lobes was therefore removed. A drainage tube was inserted.

Röntgenographic examination carried out on May 4 showed a fairly large pneumothorax. The pleural cavity was punctured and about 75 c.c. of sero-anguinolent fluid and some air were evacuated.

On May 10 the drainage stopped functioning whereupon a Büllau drainage tube was inserted. Now the lung re-expanded, but on May 17 when the patient was discharged a small pneumothorax persisted. This was subsequently replaced by a localized exudate which was evacuated.

Röntgenographic examination carried out in December, 1947, revealed increased exudate. Pleural puncture showed that the chief portion was organized, it being possible to evacuate only a few c.c. of hemorrhagic fluid.

Other methods of treatment have been attempted, but like the production of chemical pleuritis these methods seem quite insufficient from a surgical point of view. Electrocoagulation of the aperture of the fistula might produce healing in some cases but will not remove the cause of the disease (This method of treatment was applied unsuccessfully in our Case 6). The same holds good of scarification of the pleura (Churchill) and simple suture of the fistula (Movitt and associates) either of which requires fairly extensive thoracotomy.

PULMONARY RESECTION IN THE TREATMENT OF SPONTANEOUS PNEUMOTHORAX (FIVE CASES)

The development of thoracic surgery has made a more radical therapy possible. Thus, Tyson and Crandal as well as Hoyer and Clagett have successfully performed resection of pulmonary cysts in spontaneous pneumothorax. In our material pulmonary resection was performed in five cases with beneficial results.

CASE 4 (Figs. 3 and 4).—K. N., a girl, aged 21 years, was admitted to the hospital on Dec. 12, 1946 and discharged on Jan. 11, 1947.

In August, 1947, for unknown reasons, the patient suddenly experienced violent pain in the left side of the chest accompanied by dyspnea. Conservative treatment for six weeks proved ineffectual. Since then great functional dyspnea persisted and the patient was, therefore, admitted to the Department of Thoracic Surgery of the Rigshospital on December 12.

Roentgenographic examination revealed total left-sided pneumothorax with no displacement of the mediastinum. Thoracoscopy showed an emphysematous bleb about the size of a grape in the lingular area—no other abnormalities were observed.



Fig. 3.

Fig. 4.

Figs. 3 and 4 (Case 4).—Roentgenograms obtained immediately before and twelve months after pulmonary resection. In Fig. 3 a moderately great left-sided pneumothorax is seen. Fig. 4: 12 months postoperatively the roentgenogram reveals complete re-expansion of the lung with distinct pleural coverings in the sinus phrenico-costalis. There is evidence of partial regeneration of the removed rib.

On Dec. 27, 1946, thoracotomy was carried out with resection of the affected area which proved to be more extensive than revealed by thoracoscopy. Postoperative course was uneventful.

On follow-up examination on Jan. 24, 1948, the patient was in good health. Roentgenography showed slight basal pleural opacity, otherwise no abnormalities.

CASE 5.—E. H. P., a girl, aged 29 years, was admitted to the hospital on June 4 and discharged on July 5, 1947.

In 1941 she had left-sided pleuritis. In 1942, she had an appendectomy complicated by pneumonia. Otherwise there was no history of previous disease.

While walking against the wind, in October, 1944, the patient suddenly experienced violent pain in the chest accompanied by great dyspnea. The patient was admitted to a hospital elsewhere and a diagnosis of great left-sided pneumothorax was established. From this date up to the date of admission the patient experienced five similar attacks.

On May 11, 1947, the patient experienced a recurrent attack of the usual nature and was admitted to the Department of Thoracic Surgery of the Rigshospital on June 4. Daily aspiration of about 500 c.c. of air was ineffectual. Subsequent roentgenographic examination revealed a cyst in the upper lobe about the size of a walnut.

On June 12 thoracotomy was performed. Three cysts were observed, one about the size of a walnut and two smaller ones at the top of the apex. Some adhesions extending

from the cyst to the thoracic wall were cut and the affected area resected. Postoperative course was uneventful.

On follow-up examination on August 11 the patient was found to be in good health. Roentgenography showed complete re-expansion of the lung.

CASE 6.—K. L., a girl aged 20 years, had from childhood a tendency to become dyspneic.

Roentgenographic examination carried out nine months prior to hospitalization revealed no abnormalities. (A subsequent check of the x-ray plates, however, disclosed a small cyst in the upper lobe.) Six months prior to hospitalization the patient suddenly experienced slight pain in the right side of the chest. At that time the presence of a moderately large pneumothorax was discovered.

At the time of hospitalization the patient suffered from slight dyspnea, otherwise no particular symptoms. Roentgenographic examination revealed a small pneumothorax in the upper part of the lung, whereas the lower part was found to be adherent to the thoracic wall.

On August 21 thoracoscopic examination revealed the presence of a large pulmonary cyst in the right upper lobe. At the top of the cyst there was a small aperture the surroundings of which were electrocoagulated. The smoke resulting from this was blown in and out through the hole. A drainage tube ending under water was inserted.

In the following days the temperature rose from 40 to 41° C. Following penicillin therapy the temperature gradually returned to normal, but as the pneumothorax persisted, thoracotomy was performed on September 9, with resection of the cyst which was adherent to the thoracic wall through cordlike adhesions. The detached part of the cyst was removed. A small bronchus was seen at the bottom. The side walls were electrocoagulated and sutured.

On follow-up examination twelve months later there were no complaints. Roentgenography showed small pleural coatings over the apex and the diaphragm, otherwise no abnormalities.

CASE 7.—I. H., a woman aged 30 years, was admitted to the hospital on Dec. 5, 1947, and discharged on Dec. 22, 1947.

Some day in June, 1947, the patient suddenly experienced heavy pain in the left side of the chest accompanied by great dyspnea. She was admitted to a hospital elsewhere. Here the patient was found to be affected with a total left-sided pneumothorax with displacement of the heart and mediastinum. Following repeated aspirations the patient became symptom free.

Two weeks prior to admission to the Rigshospital the patient experienced a recurrent attack of the same nature. At the time of hospitalization the patient was suffering from slight dyspnea. Roentgenographic examination revealed a total pneumothorax with a small exudate in the sinus.

Thoracoscopy was carried out on December 8. A thin-walled emphysematous bleb about the size of a cherry was observed in the upper lobe anteromedially. The lower lobe was atelectatic.

On December 12 thoracotomy with resection of the affected area was performed. In this location there was a cyst having a pedicle about 2 cm. wide. A number of cordlike adhesions extended from the cyst to the thoracic wall. The cyst was resected.

The portion removed was easily reinflated through a small bronchus and now another small cyst became visible on one side of the inflated vesicle. In this cyst there was a small slit which was closed by a clamp. Thereupon it was possible to inflate also this cyst through another small bronchus. It was impossible to reexpel the air by pressure of the fingers. There is, therefore, no doubt that in this case there existed a valvular system in which the valvular vesicle ruptured. On follow-up examination on Jan. 7, 1948, the patient stated that she was in good health. Roentgenographic examination revealed complete re-expansion of the lung.

CASE 8.—C. G. H., a man aged 39 years, was admitted to the hospital on Dec. 31, 1947, and discharged on Jan. 10, 1948.

On Dec. 20, 1947, while working, the patient suddenly experienced violent, shooting pain in the middle of the thorax and the abdomen, accompanied by heavy dyspnea and cough. The physician first attending the patient regarded the case as a lesion of the abdominal rectus. The patient was referred to the surgical polyclinic of the Rigshospital where the presence of a large pneumothorax was detected and the patient was hospitalized.

Roentgenographic examination revealed total right-sided pneumothorax with no displacement of the mediastinum. In the upper lobe a net like shadow was distinctly observable which suggested the presence of cysts in this location. Thoracoscopy was not carried out owing to the positive roentgenographic findings. On Jan. 2, 1948, thoracotomy was performed with lobectomy of the right upper lobe. Superoposteriorly in the upper lobe an area the size of an orange was seen covered with numerous vesicles of varying size; anteriorly there was an area containing minute densely grouped cysts. Along the lower boundary there was a string of fine emphysematous blisters resembling tiny beads. Segmentary resection was first attempted, but had to be given up, wherefore lobectomy was carried out. A drainage tube with permanent suction was inserted.

Roentgenographic examination carried out on January 6 revealed a small pneumothorax over the apex with reflecting liquid. Following pleural puncture 25 c.c. of clear liquid and 60 c.c. of air were withdrawn.

On follow-up examination on February 9, the patient was feeling well, Roentgenographic examination revealed that a small pneumothorax, about the same size as detected by examination on January 12, persisted over the apex.

Our experience with operative treatment of the disease has been favorable, the follow-up examinations having shown subjective well-being of the patients and the roentgenological changes having been only slight. As a rule we observed only obliteration of the sinus angle or small localized coatings of the pleura. In one case a small pneumothorax persisted one month after the operation, but from experience gained in other lobectomies it is safe to assume that spontaneous resorption will take place.

Surgical intervention was indicated by chronicity in two cases (Cases 4 and 6), by recurrence of attacks in two cases (Cases 5 and 7). In one case roentgenographic examination revealed the presence of cysts and was thus suggestive of an extensive process (Case 8). It is our impression that the patients receiving operative treatment do not have to stay in bed for so long a period as those receiving silver nitrate injections. The five patients operated on by us left the hospital on the thirteenth, fourteenth, fifteenth, twenty-third, and thirty-third postoperative days, respectively. In the last case special circumstances prevailed. Patients receiving conservative treatment exclusively frequently have to stay in the hospital or in a sanatorium for several months.

CONCLUSION

The present status of thoracic surgery makes it possible at practically no risk to undertake radical treatment of spontaneous pneumothorax aiming at complete extirpation of the affected area. Such treatment is especially indicated in cases where chronicity develops or where recurrent attacks or tension pneumothorax occurs.

While medical treatment, naturally, does not remove the actual cause of simple spontaneous pneumothorax, that is, the cysts or the vesicles, roentgeno-

graphic and especially thoracoscopic examination are valuable aids in establishing a correct diagnosis and locating the process which, in turn, makes surgical treatment possible.

Experience confirming the theoretical views shows that the safest surgical treatment is complete resection of the affected area. The other more conservative methods, such as the production of chemical pleuritis, electrocoagulation, or simple suture of the fistula, do not fulfill the requirements, namely, complete recovery, as they do not preclude the possibility of recurrent attacks.

In cases where it is likely that adhesions will bring about rupture of the visceral pleura resulting in pneumothorax, cauterization of adhesions will undoubtedly constitute sufficient therapy.

SUMMARY

A survey is given of the literature with special reference to etiology and diagnosis. Routine thoracoscopy is recommended as a valuable aid in establishing the correct diagnosis and locating the site of the disease in cases where roentgenographic examination is negative.

In our series of eight patients thoracoscopy was carried out in six cases. In five of these cases cysts were detected in the upper lobe, in one case a presumably ruptured subpleural vesicle was observed at the base of a wide adhesion.

Three of our eight patients received treatment by the method of Brock, 1 c.c. of silver nitrate in 10 per cent solution being injected. In two of these cases results were satisfactory, whereas in one case a fairly great organized exudate was seen over the top of the lung with marked displacement of the trachea.

In five cases resection of the affected area was carried out successfully. One month postoperatively a small pneumothorax over the apex of the lung was observed in one case, the remaining cases revealed no abnormalities apart from insignificant pleural opacity.

The latter method is recommended as being the most rational, whereas the silver nitrate method of Brock should be reserved for debilitated and elderly individuals whom it would not be advisable to expose to major surgery, and for cases of multiple cysts.

REFERENCES

- Abbot, T. R., and Renault de Oliveira, H.: *J. THORACIC SURG.* 14: 453, 1945.
Andersen, E. W.: *Nord. med.* 35: 1703, 1947.
Babington, S. H.: *West. J. Surg.* 52: 73, 1944.
Bethune, N.: *J. THORACIC SURG.* 4: 251, 1935.
Brock, R. C.: *Guy's Hosp. Reports* 91: 99, 1942.
Castex, M. R., and Mazzei, S.: *Arch. med-chir. de l'app. respir.* 12: 23, 1937.
Churchill, E. D.: Personal communication referred by Tyson and Crandall, *J. THORACIC SURG.* 10: 566, 1941.
Cohen, S., and Kinaman, J. M.: *New England J. Med.* 235: 461, 1946.
Cutler, J. W.: *J. A. M. A.* 111: 408, 1938.
Emerson, C. P., and Becker, R. C.: *Am. J. Roentgenol.* 10: 126, 1923.
Fischer, R. L.: *Ann. Int. Med.* 4: 1395, 1931.
Gordon, I.: *Lancet* 2: 178, 1936.
Grapengiesser, S.: *Acta med. Scandinav.* 85: 505, 1935.
Gullbring, A.: *Nord. med.* 29: 307, 1946.
Güttsche, C.: *Ugesk. f. læger* 95: 765, 1933.

- Hamman, L.: *Ann. Int. Med.* 13: 923, 1939.
- Hemmel, H.: *Arch. Int. Med.* 63: 649, 1939.
- Hoyer, C. P., and Clagett, O. T.: *J. THORACIC SURG.* 15: 418, 1946.
- Horsley, J. S., and Bigger, I. A.: *Operative Surgery*, St. Louis, 1937, The C. V. Mosby Co. p. 605.
- Kjaergaard, H., *Spontaneous Pneumothorax in the Apparently Healthy*, Copenhagen, 1932, Levin & Munksgaard.
- Kjaergaard, H.: *Acta med. Scandinav.* 80: 93, 1933.
- Kulke, E.: *München. med. Wehnschr.* 67: 1175, 1920.
- Locke, E. A.: *M. Clin. North. America* 13: 75, 1929.
- Macklin, C. C.: *Canad. M. A. J.* 36: 414, 1937.
- Macklin, C. C.: *Arch. Int. Med.* 64: 913, 1939.
- Macklin, C. C., and Macklin, M. T.: *The Herbert Evens Birthday Volume*, University California Press, page 335, 1943.
- Macklin, C. C., and Macklin, M. T.: *Medicine* 23: 281, 1944.
- Mainini, C., and Alvarez, A.: *Prensa méd. argent.* 22: 1411, 1935.
- Mourier, P.: *Ugesk. f. laeger* 99: 1023, 1937.
- Movitt, E. R.: *Am. J. M. Sc.* 209: 595, 1945.
- Movitt, E. R., Smith, J. V., and Elsener, S.: *Dis. of Chest* 13: 221, 1947.
- Ornstein, G. G., and Lercher, L.: *Quart. Bull., Sea View Hosp.* 7: 149, 1942.
- Orrego Puelma, H., and Richard, B.: *Prensa méd. argent.* 32: 1251, 1945.
- Ross and Fullerton: *National Research Council Office of Medical Information*, Washington, Dec. 1944. (Referred by Warming, J. J.)
- Schneider, L., and Reissman, J.: *Am. J. Roentgenol.* 54: 12, 1947.
- Schoner, A., and Erlich, D. E.: *M. Bull. Vet. Admin.* 11: 206, 1935.
- Singer, J. J., Jones, J. C., and Tragerman, C. J.: *J. THORACIC SURG.* 10: 251, 1941.
- Spengler, L.: *Schweiz. med. Wehnschr.* 4: 309, 1923.
- Sycamore, L. K.: *Am. J. Roentgenol.* 36: 844, 1936.
- Trowbridge, N.: *Arch. Int. Med.* 73: 460, 1944.
- Tyson, D., and Crandal, W. B.: *J. THORACIC SURG.* 10: 566, 1941.
- Warming, J. J.: *National Research Council of Medical Information*, Washington, Dec., 1944.
- Watson, E. E., and Robertson, C.: *Arch. Surg.* 16: 431, 1928.
- Wilson, J. L.: *Internat. Clin.* 1: 157, 1937.
- Wood, H. G.: *Minnesota Med.* 14: 550, 1931.

THE ANESTHETIC BLOCKING OF THE VAGUS, THE SYMPATHETIC AND THE PHRENIC AT THE NECK IN THORACIC SURGERY— ITS PROTECTIVE ACTION ON BLOOD PRESSURE

EXPERIMENTAL RESEARCHES

FRANCO SOAVE, M.D.

GENOA, ITALY

DURING the ten years from 1938 to 1948 a considerable step forward has been made in thoracic surgery, as is witnessed by the large number of operations successfully carried out and duly checked in the process of time, showing the high degree of technical perfection reached by the surgical cure of endothoracic diseases. An important contribution has been offered by the constant improvements in narcosis by hyperpressure, while a further improvement has been obtained through the realization of controlled respiration, enabling the overcoming of respiratory difficulties caused by an opening in the thorax. By way of curarization it is now possible to obtain complete muscular relaxation of the patient, even when using a moderate dose of anesthetic.

Through this particular form of narcosis it is possible to maintain, in all circumstances, an efficient respiratory exchange, even during the eventual opening of both pleural cavities. Thus, while apnea was formerly cause of complications and death, today, with controlled respiration narcosis, it can always be obtained when complete immobilization of the operative field is required. It is, however, advisable during endothoracic interventions on the lung, not to reach a total collapse of the healthy lobes of the lung on the operated side, for a complete collapse, extending for a fairly long time, might prevent re-expansion in the period immediately following operation (Meier). The opening of the thorax nullifies the negative pressure essential to the proper filling of the heart, with the result that a smaller quantity of blood reaches the lung. By using an endotracheal tube which enables the respiratory muscles to be set at rest, this complication may be relieved, but it is impossible to avoid the increase of endopulmonary pressure with its consequent increase of respiratory resistance. Though by controlled respiration and by the use of curare a substantial improvement has been reached, yet hitherto it has been impossible to conquer the numerous nervous reflexes caused by operative maneuvers on endothoracic, mediastinal and hilar tissues, resulting in cardio-circulatory depressions, as was demonstrated even recently by Biancalana, Dei Poli, and Soave in intubated and curarized animals.

The pathogenic mechanism of such phenomena, in which the reflex nature appears certain, had been the object of study by a large number of experimenters. Yet the problem of the presence of cardiocirculatory and respiratory disturbances caused by operative maneuvers on the vagosympathetic plexus remains unsolved.

From the Department of Surgery, Genoa University (Dir. Prof. L. Stropeni).
Received for publication June 24, 1948.

In order to give a clearer insight of the problem which I am endeavoring to examine, I shall submit a few remarks on lung innervation.

The vagus nerve of the right hemithorax, after having given rise to the recurrent nerve, gives off the thoracic branches divided into tracheal, bronchial, esophageal, pericardial, and infracardial.

Morin and Dei Poli include among the thoracic branches the "middle cardiac" which they have often noticed arising from the vagus nerve, below the recurrent nerve, while other authors have found these nerves actually to originate from the recurrent nerve.

The anastomosis of the cardiac nerves of the vagus with those of the sympathetic-thoracic plexus cannot be definitely set out, owing to their variability, within the frame of a definite scheme (Hovelacque); as a rule they originate from the stellate ganglion or from the second and third thoracic ganglia. These vagus-sympathetic anastomoses among cardiac nerves of the two systems have a great importance, for through them the lung is reached by a considerable complex of sympathetic fibers going in a small portion to the anterior pulmonary plexus. The larger and more conspicuous group of pulmonary nerves detaches itself from the vagus in the section where the latter crosses the posterior part of the bronchus. From these nerves originate capillary branches, partly innervating the bronchus and partly innervating the pulmonary parenchyma and the vessels. The above branches, which have been described as belonging to the vagus, contain a sympathetic component proceeding from vagosympathetic anastomoses of the upper portion of the thorax. The bronchopulmonary branches properly anastomosed constitute the posterior pulmonary plexus and are particularly laid in proximity of the reflection line of the mediastinal pleura on the lung root; while extending toward the hilus, a few fibers pass over the bronchus, placing themselves on its ventral aspect. Thus, the bronchus, in its segment close to the pulmonary hilus, is mainly covered by bronchopulmonary nerve ramifications. On the posterior aspect of the root run the bronchial arteries accompanied by their nervous equipment, the filaments of which extensively anastomose themselves with those of the vagus. In the left portion of the hemithorax the nerve distribution and topography are mainly similar to those of the right hemithorax. It is, however, important to lay stress on the fact that, as already demonstrated by Taft, the pneumocardial branches of the left hemithorax split themselves at a higher level and are surrounded by loose connective tissue, without establishing a too close connection with the vessels and the bronchi, while on the right side the same are rather close to the trachea. At a later stage Taft gave much importance to this anatomic display in order to explain the greater danger of pulls on the right pulmonary root noticed by Sauerbruch, Graham, and by Denk in human surgery, and experimentally demonstrated by Kusakabe. The latter, by electric stimulation of the right pulmonary root of a dog, noticed a more distinct slackening of pulsation and respiration together with a lowering of blood pressure than by stimulating the left pulmonary root.

From the foregoing brief anatomic premises it follows that the functions of the cardiac and pulmonary vagus are likely to be reciprocally influenced.

The cooling of these nerves at the neck causes, indeed, a cardiac acceleration reflecting itself in respiratory changes; likewise, the electric stimulation of the central trunk of the vagus causes respiratory and pressure changes proportionate to the concentration and the duration of the stimulations.

Stimulations of weak concentration produce an increase of respiration frequency and a decrease in the size of the thorax when in an inflationary position; an increase in stimulating power still increases these effects; when the frequency and the intensity become greater, respiration stops in an inflation position which may be turned into a deflation position through stimulations of a still greater intensity.

The effects on pressure are even more changeable: light stimulations produce at first a lowering of pressure (Hunt); increased stimulation decreases depressing effect; much stronger stimulation increases pressure proportionate to its frequency and duration. When the increase of pressure is not very strong, the interruption of stimulation produces a decrease of pressure and a respiratory acceleration.

The pressure effect of stimulation may be transformed into a contrary effect by the action of anesthetics (Cyon) or by the cooling of the nerve (Hunt).

The reason for this variable response has been attributed to the presence of stimulating and depressing fibers both in the vagus (Heymans, Mac Osvallo, Tournade) and the other sensory nerves (Hunt).

Cannon and Rapport attribute the lowering of pressure to reflex inhibition of adrenalin secretion (adrenergic-sympathetic fibers).

Though the functions of the sympathetic nerve are known in their broad lines, they are still scarcely known in their mechanical reflexogenic endothoracic details.

Though attributing to the vagus and the sympathetic nerves the greatest importance in the determination of reflexes on blood pressure and respiration, one should pay particular attention to the diaphragmatic receptors sending their impulses to the center and to the periphery by way of the phrenic nerve.

In natural respiration there are discharges of impulses synchronizing with inflation and proportionately larger as respiration becomes more energetic. In apnea caused by hyperventilation the nerve rests but becomes active again when aspiration is made in the trachea or when the abdomen is so compressed as to push the diaphragm toward the thorax (Cardin).

Discharges of impulses are caused even when a pull in cephalic direction is made on the tendinous center of the animal's diaphragm when the thorax has been opened (Cardin). In respect of tension, of change of circulatory and respiratory conditions, and of blood gases, these diaphragmatic tensoreceptors behave in exactly a similar way as the tension proprioceptors of the pulmonary vessel (Adrian) and of the carotid sinus (Matthew and Brouk). It is generally admitted that the rhythm and the depth of respiration are governed by nervous impulses coming from the lung (Hering-Breuer) and reaching the respiratory center through the vagus (Adrian).

Electric stimulations of the afferent discharges directed toward the center from the pulmonary fibers of the vagus show that during normal respiration rhythmical impulses are determined only during inflation, and the impulse frequency in each single fiber is in relation to the entity of distention of the lung and is not liable to large variations until that particular stimulation lasts. By varying the stimulation, respiratory modifications can be favored and be made to influence blood pressure. This shows that pulmonary receptors are not liable to a prompt adaptation, yet in tracheotomized animals balancing and adaptation reflexes have been noticed to set in, in order to re-establish a proper pressure balance.

Head, Hommonda, and Wilson explain such balancing reflexes by admitting the presence of a further order of afferent fibers interested in Hering-Breuer reflexes. Such fibers displayed their action more especially during pulmonary collapse, as was demonstrated during experiments made by extracting air from the lung, and also during a respiratory spasm in order to compensate for an obstruction (Whitteridge). It seems that the coordinated activity of these two fiber systems regulates the rhythm of respiration, thus harmonizing the inflation and deflation mechanism.

Surgical experience past and present shows that thoracotomy by itself, pleural, endothoracic, pericardial, and diaphragmatic stimulation, may cause hypotensive reflexes, but it is commonly agreed today that such reflexes mainly originate by direct stimulation of the hilus and the pulmonary root. In these hilar areas surgeons endeavor to attenuate the vagal-sympathetic tonus by using Novocain infiltrations of the pulmonary hilus and its elements.

O'Shaughnessy observed that respiratory alterations particularly follow faradic stimulation of the posterior pulmonary root, owing to the reflexes generated in the posterior pulmonary plexus unusually rich in centripetal vagal branches belonging to the bronchial nervous system. By stimulating the anterior aspect of the root, and more especially the pulmonary veins, O'Shaughnessy noticed a modification of the cardiac rhythm and a fall of blood pressure. This author believes that fibers running near the pulmonary veins originate in the deep cardiac plexus and have no connection with the vagus trunk. In conclusion, the fall of pressure would be due to a local reflex following direct irritation of the nerve center of the heart.

According to Bross and Lueken pressure falls are particularly due to the bronchoconstrictive action of the vagus.

Bilfeldt-Nicholls and Escala Bawden (1946) give much importance to mediastinal fluctuation and to mechanical impediment of the blood flow into the heart in order to explain the origin of the serious hypotensive reflexes. Morin and Dei Poli believe that a portion of reflexes capable of producing more noticeable changes of blood pressure are exclusively due to stimulation and pulling of the pulmonary veins; they attach great importance to the sensitive receptors of the subendothelial stratum of the pulmonary veins.

The researches made by these authors justify the necessity of Novocain infiltration of the hilus and of the pulmonary root.

Today, with controlled respiration narcosis, it is possible to harmonize, even with a wide-open thorax, pulmonary rhythm and thus eliminate a portion of the causes likely to discharge hypotensive reflexes caused by sudden modifications of pulmonary ventilation.

With the use of curare, relaxation of the respiratory and intercostal muscles, and even of the diaphragm, can be obtained if so required. Yet even by this technique vagal and diaphragmatic depressive reflexes are not abolished.

In March, 1947, Parenti submitted to the Societa' Piemontese di Chirurgia the favorable results obtained by him in thoracic surgery during World War II by a simultaneous anesthesia of the vagus, of the sympathetic, and of the phrenic at the neck. According to him, by this anesthesia pleural shock, respiratory collapse, and cough are attenuated, while operating conditions and those of the patient are improved.

Starting from Parenti's observations, I made the object of my researches not merely to check the practical advantage of his suggestion, but also to verify whether by this technique it is possible to attenuate hypotensive reflexes not merely arising from the pleura, as in accordance with the author's premises, but originating above all by surgical maneuvers of the hilus and of the pulmonary root.

Such reflexes are particularly dangerous in vagotonic subjects and cannot be completely suppressed by Novocain infiltrations in the pulmonary hilus (Soave). In everyday practice, while anesthesia of the sympathetic and alcoholization of the phrenic are used for therapeutic aims, it does not appear that the simultaneous anesthesia of the three nerves (vagus, sympathetic, and phrenic) as studied by me has yet been applied.

TECHNIQUE

The experiments were made on twenty dogs of robust constitution duly anesthetized with a solution of chloralose, 5 ctg. every kilogram weight, and urethane, 35 ctg. every kilogram weight, administered by intravenous injections, the operating room being kept at a constant temperature, pressure, and moisture. Blood pressure during the whole experiment was registered by connecting a Ludwig manometer to the femoral artery. Time spent on the operation was registered by a tonometer. Magnesium sulfate was used to prevent clots from obstructing blood pressure readings.

Each animal, after general anesthesia, was submitted to artificial respiration by way of a Palmer pump and the chest cavity was subsequently opened after the removal of a rib. All experiments were made on the right hemithorax for the reasons just mentioned.

Through the lateral opening and using a curved Klemmer with long and thin tongues I have carried out a number of mechanical compressions and pulls of the lung root in its entirety, of the lung veins, and of the pulmonary artery alone. Such mechanical compressions, stimulations, and pulls have been exerted in all animals before and after anesthetic block of the three nerves (vagus, sympathetic, and phrenic) at the neck.

The injection was made with a very thin needle, using a 0.50 per cent solution of Novocain, without adrenalin, in doses of 10 to 20 c.c. After having pulled aside with a finger the middle margin of the sternocleidomastoid, the same finger was used to shift the common carotid and the exterior jugular vein. By this maneuver the vagus, which lies at the top corner formed by these two vessels, is also shifted and has a tendency to superimpose itself on the sympathetic cord. Having thus externally deviated the vessel bundle and always keeping the vessels well aside, the needle is deeply inserted until it reaches the stratum of the deep neck muscles, and in this area (lower margin of the larynx) the anesthetic infiltration is applied.

It is thus possible to obtain vagus and sympathetic anesthesia. In order to reach the phrenic one must push forward the needle point for approximately $\frac{1}{4}$ cm. Then, sliding on the prevertebral fascia above the anterior scalene, the needle is in the immediate vicinity of the phrenic.

The complete protocols of the experiments carried out by the author are not given here. Only diagrams of some experiments are reported.

The experiments carried out by me have confirmed that:

(1) In normal animals crushing of the lung root in its entirety causes a considerable drop of blood pressure which becomes accentuated if stimulation is made by pulling.

(2) Crushing of pulmonary veins is followed by a drop of blood pressure considerably lower than that caused by the crushing of the entire root. If lateral or inferior stimulation by pulling is added to vein crushing, a more pronounced blood pressure takes place.

(3) Crushing and pulling of the pulmonary arterial branch causes less pronounced alterations of blood pressure.

The hypotensive reflexes caused by surgical maneuvers have generally been proved to be reversible, and the level of blood pressure has been established in a more or less constant lapse of time (in seconds) for all animals.

In all animals treated with the simultaneous block of the vagus, the sympathetic, and the phrenic at the neck under the same experimental conditions, I have constantly noticed that hypotensive reflexes were always present, but much less pronounced and more rapidly reversible.

The aim of my study was to establish, through a scientific demonstration, whether this anesthetic block attenuates, better than Novocain infiltration of the root, the alterations of blood pressure caused by manipulations of pulling or crushing of the lung root after the chest cavity has been opened.

I believe that the block at the neck, by inhibiting a large portion of the phrenic, eliminates the diaphragm's spasm and diminishes reflexes originating in diaphragmatic tensorceptors which trouble blood pressure; in the second instance it is likely that simultaneous anesthetic action of the vagus and the sympathetic may considerably mitigate reflexes proceeding from the root. It is easy to notice this in Figs. I to VII. I have always exerted the pulling in such a way as not to cause a shifting of the mediastinum, in order to avoid the production of hypotensive reflexes due to the indirect pull of the nervous plexi of the hemithorax.

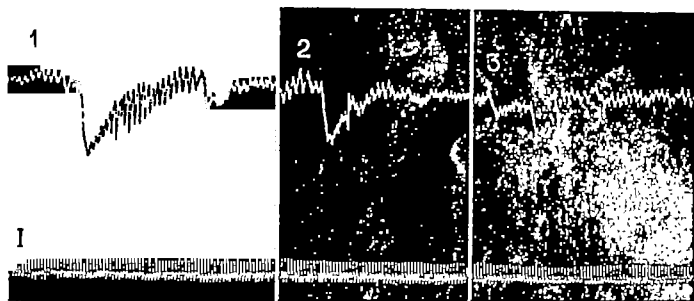


Fig. I (Experiment 1, Trial 1, Control).—1, Crushing and pulling of lung root in its entirety; 2, similar maneuver on pulmonary vein; 3, similar maneuver on pulmonary artery. (Figs. 1 to VII from Chirugla, Milano, No. 5-6 1918.)



Fig. II (Experiment 1, Trial 2).—After twenty minutes from the previous trial the block of the three nerves is exerted at the neck. 1, Crushing of the root; 2, crushing of the bronchus; 3, after ten minutes, crushing of pulmonary vein; 4, crushing of pulmonary artery; 5, crushing and pulling of the bronchus.

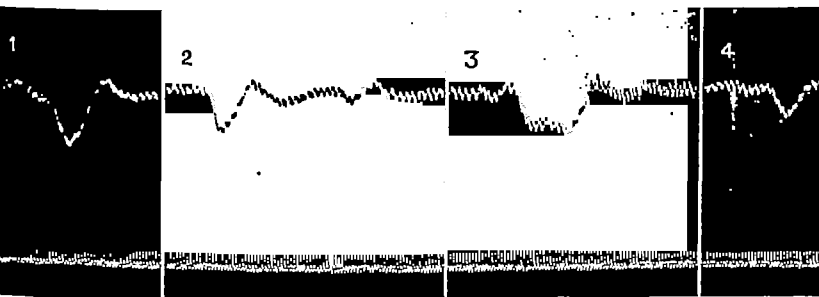


Fig. III (Experiment 2, Trial 1, Control).—1, Crushing and pulling of lung root; 2, after ten minutes, crushing of pulmonary vein; 3, crushing and pulling of pulmonary vein; 4, after ten minutes crushing and pulling of pulmonary artery.

REFERENCES

- Adrian, E.: Afferent Impulses in the Vagus and Their Effect on Respiration, *J. Physiol.* 79: 332, 1933.
- Beecher, H. K.: Principles of Anesthesia for Lobectomy and Total Pneumectomy, *Acta. Med. Scandinav.*, Supp. 90, p. 146, 1938.
- Biancalana, L., Dei Poli, G., and Soave, F.: On Reflexes of Pulmonar Hilus in Experimental Curarization, *Bulletin Piemontese Surgical Society* 17: 9, 1947.
- Bilfeldt-Nicholls, W. H., and Escala Bawden, A.: Role of the Pulmonary Plexus in Lung Surgery, *J. THORACIC SURG.* 15: 422, 1946.
- Bross, W., and Lueken, B.: Les troubles de la circulation du sang et du volume respiratoire au cours des operations sur les poumons et le problème du mecanisme nerveux du choc operatoire, *Rev. de Chir.* 57: 648, 1938.
- Cardin, A.: Receptors of Diaphragm's Tension, *Arch. Biol. Sc.* 30: 1, 1944.
- Hammonda, M., and Wilson, W.: Reflex Acceleration of Respiration Arising From Excitation of Vagus or Its Terminations in the Lungs, *J. Physiol.* 94: 497, 1939.
- Laughton, Hewer: Trichlorethylen as an Anaesthetic Agent, *Brit. M. Bull.* 4: 108, 1946.
- Kusakabe, T.: Experimentelle Untersuchungen der Innervation der Lungenwurzel und ihre klinische Bedeutung, *Deutsche Ztschr. f. Chir.* 238: 380, 1933.
- Morin, F., and Dei Poli, G.: On Reflexes of Pulmonar Root, *Arch. Med. Sc.* 48: 229, 1947.
- Parenti, G. C.: The Anesthetic Blocking of Vagus—Sympathicus and Phrenicus and Its Applieance in Thoracic Surgery, *Minerva chir.* 2: 89, 1947.
- O'Shaughnessy, L.: The Vagus and Its Relation to Surgery of the Lung, *J. THORACIC SURG.* 5: 386, 1936.
- Sunder, Plasmann P.: Nervous Receptors Area in Walls of Intrapulmonary Bronchi of Man, *Deutsche Ztschr. f. Chir.* 240: 249, 1938.
- Whitteridge, D., and Bulbring, E.: Change in Activity of Pulmonary Receptors in Anaesthesia and Their Influence on Respiratory Behaviour, *Brit. M. Bull.* 4: 85, 1946.

NOTICE

In June, 1949, the Secretary of the Board of Thoracic Surgery met with the American Board of Surgery, and the problem of those individuals who are neither members of The American Association for Thoracic Surgery nor diplomates of the American Board of Surgery was considered. The American Board of Surgery allowed a new group to be admitted to the Founders Group at the discretion of the Board of Thoracic Surgery. This group will be known as group "D" and will consist of other surgeons who are not members of The American Association for Thoracic Surgery or diplomates of the American Board of Surgery and whose training, because of the period in which it was taken, does not qualify them to become certified by the American Board of Surgery, but who may upon careful scrutiny and adequate evidence of proficiency be eligible for consideration as founder members.

THE failure of the collapsed lung to re-expand after the occurrence of an attack of spontaneous pneumothorax results in chronic invalidism for the patient. In certain instances the persistence of the pneumothorax had been a major factor in contributing to the fatal outcome of these cases. Yet in spite of the potential seriousness of chronic or recurring pneumothorax, very little attention has been paid to this condition in the medical literature. There have not been many cases of chronic spontaneous pneumothorax reported in which active therapy has been administered. Most of these have been treated by chemical irritants. We have been able to find only 20 cases in the medical literature in which surgical measures have been employed,¹⁻¹² 12 of which have been reported during the past year. Because the condition is primarily a mechanical one, we are convinced that in most instances surgical measures are indicated, if maximum pulmonary function is to be preserved. Certain general principles of surgical treatment have been presented in a previous communication by one of us.¹³ This report is concerned with the discussion of the factors producing the spontaneous pneumothorax and the surgical therapy employed in a series of 15 patients treated during the past ten years.

TERMINOLOGY

The term, "spontaneous pneumothorax" is used to designate that type of pneumothorax coming on suddenly in seemingly healthy persons in whom there appears to be no obvious etiology for the pneumothorax. It occurs without external force and the disease processes responsible for this condition are generally

Read at the Twenty-ninth Annual Meeting of The American Association for Thoracic Surgery, New Orleans, La., March 28-31, 1949.

Adrian, E.: Afferent Impulses in the Thorax. *Ann. Surg.* 79: 332, 1933.
 Beecher, H. K.: Principles of Thoracic Surgery. *Med. Scandinav.*
 Biancalana, L., Dei Poli, G.: Observations on the Effect of Curarization in the Lungs. *Chir. Ital.* 1934.
 Bilfeldt-Nichols, S.: Where Direct Incision is Indicated. *Ann. Surg.* 1934.
 Bross, I. D. J.: On the Pathogenesis of Spontaneous Pneumothorax. *Am. J. Surg.* 1934.
 The pathologic lesion in spontaneous pneumothorax is a cyst, bleb, or bulla. The rupture of the

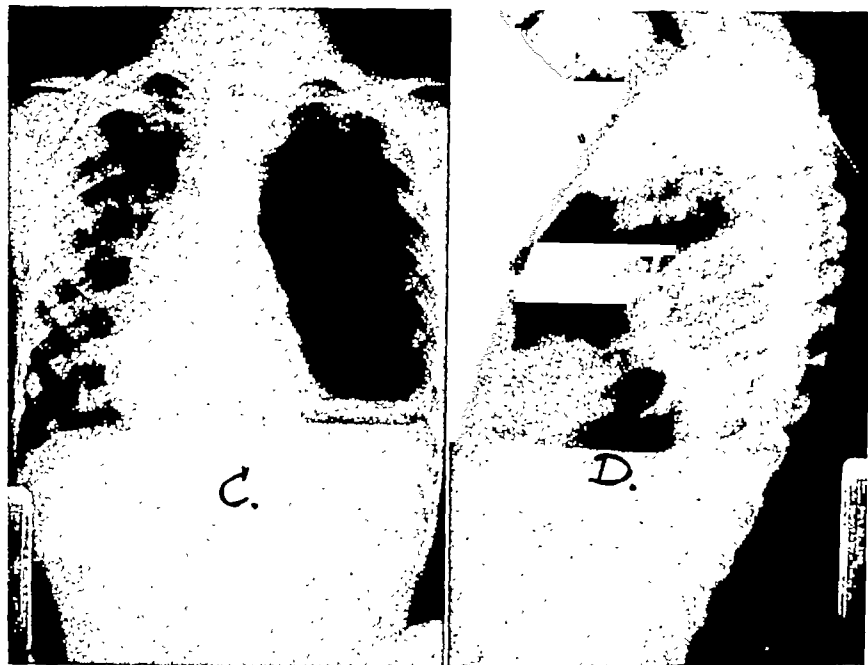
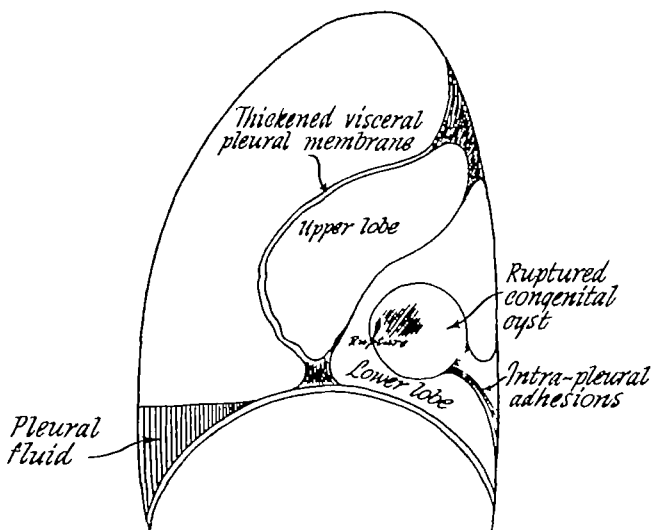
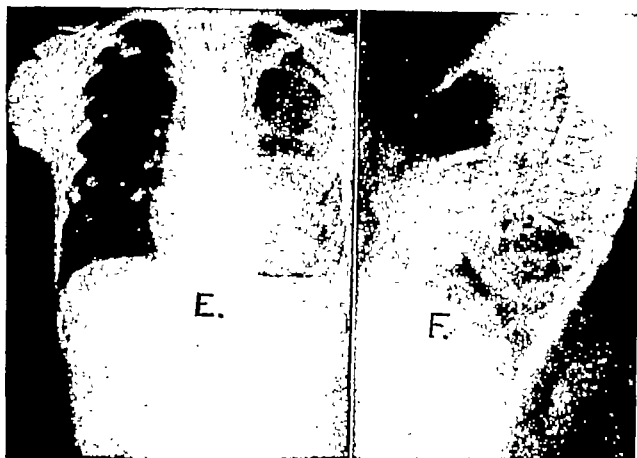


Fig. 1, A to G (Case 1).—A 6-year-old boy complained of dyspnea and pain in the left chest of six months' duration. In spite of repeated attempts to expand the lung, it remained chronically collapsed. Following open thoracotomy with resection of the ruptured congenital cyst, pneumonolysis of the lung, and pulmonary decortication, rapid expansion of the lung was obtained.

Fig. 1, A and B.—A, Roentgenogram taken immediately after onset of attack of spontaneous pneumothorax showing shift of mediastinum, depression of diaphragm, and dense adhesion between left lower lobe and chest wall. B, Temporary partial expansion of lung after insertion of needle attached to underwater drainage system.

Fig. 1, C and D.—Left lung 85 per cent collapsed with shift of mediastinum to the right. Compression changes right lung. Left lung compressed posteriorly on lateral film. Marked thickening of visceral pleural shadow. Fluid in left pleural cavity to the dome of the diaphragm.

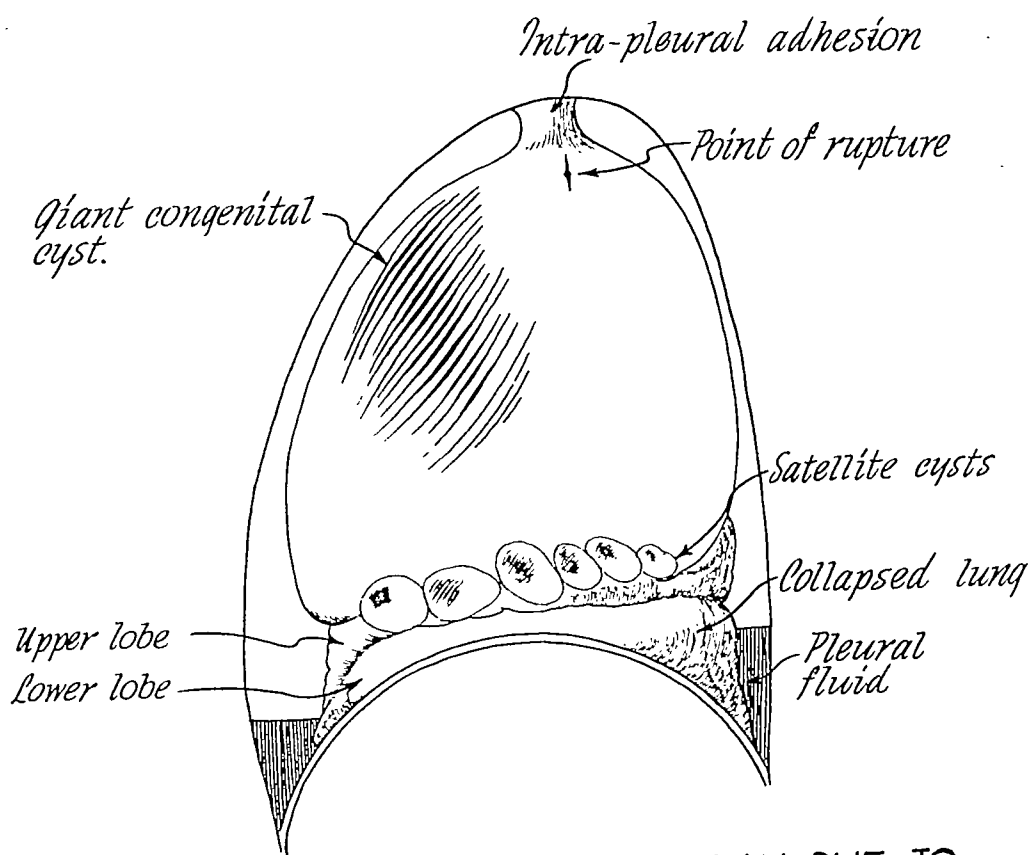


SPONTANEOUS PNEUMOTHORAX DUE TO RUPTURED CONGENITAL CYST.

G.

Fig. 1. E and F.—E, Posteroanterior view of chest two months following operation. The lung is completely expanded. F, Lateral view of chest. Note clearness of costophrenic angle. Fluoroscopy shows that the left diaphragm moves freely.

Fig. 1. G.—Drawing of the findings at operation. Lung greatly collapsed and held firmly by an inelastic visceral pleural membrane. Ruptured congenital cyst is held on tension by a dense intrapleural membrane. Pleural fluid is present. Rapid pulmonary expansion followed decortication of the lung, resection of the pulmonary cyst, and complete pneumonolysis.



SPONTANEOUS PNEUMOTHORAX DUE TO RUPTURED CONGENITAL CYST

C.

Fig. 2, A to C (Case 2).—A 17-year-old schoolboy had severe dyspnea for three months following an attack of left-sided pain. Attempts to expand the lung were unsuccessful. Weight loss of fifteen pounds.

Fig. 2, A and B.—A, Roentgenogram taken three months after onset of pneumothorax. Small pneumothorax left. Giant cyst of left upper lobe of lung held out by fine adhesions. Shift of the mediastinum to the right. Compression changes, right lung. Small amount of fluid left costophrenic angle. B, Film showing complete expansion of the lung. Patient well without recurrent attacks seven years after thoracotomy. He played tennis and has no physical impairment.

Fig. 2, C.—Small pneumothorax pocket present. A giant congenital cyst of left upper lobe is held up by dense adhesion at apex. Smaller satellite cysts are seen. Pleural fluid is present. Site of rupture of cyst demonstrated only on covering cyst with water. The lung expanded rapidly following the resection of the congenital cyst.

permanent. It is not our purpose to consider the many factors responsible for the production of emphysema. The most important are chronic or repeated pulmonary, bronchial, and bronchiolar infection, disturbance in the pulmonary circulation, and structural deformities of the chest wall and lung (Mayer and Rappaport²⁰). With the destruction of pulmonary alveolar septa, and the thinning out of the alveolar walls, we have a "weakened point" in the lung. It would be expected that rupture of these bullae would be common. However,

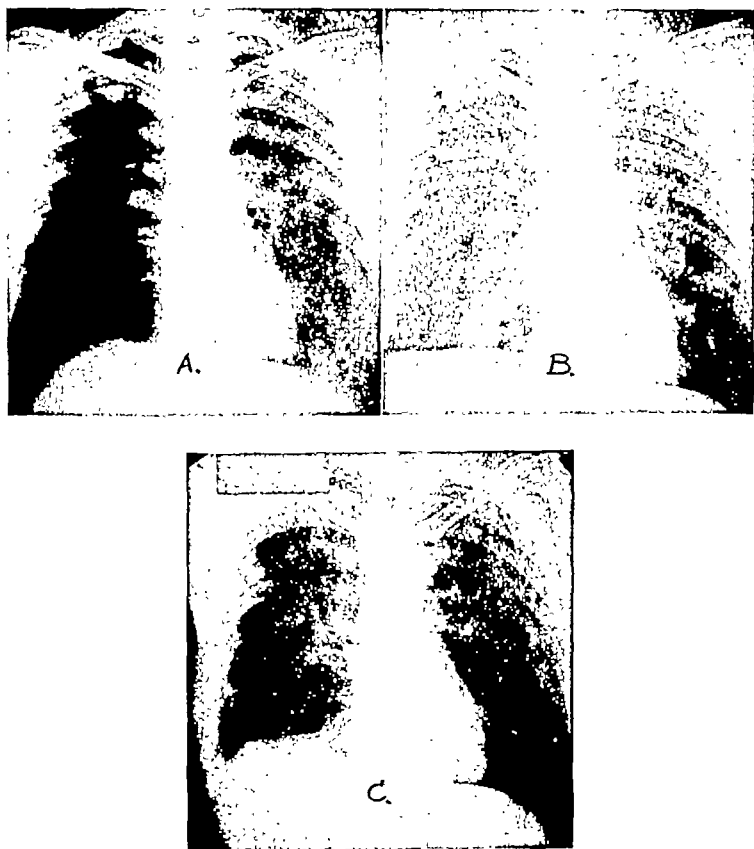


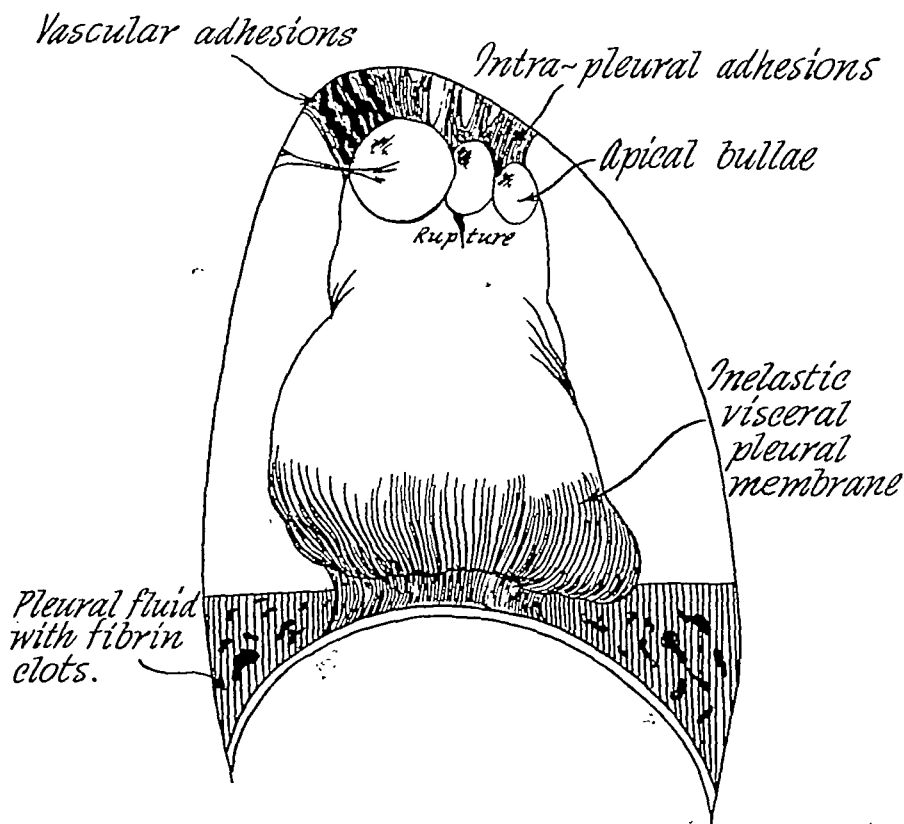
Fig. 3, A to C (Case 3).—A 31-year-old man had severe dyspnea and right chest pain for three months. The diagnosis of spontaneous pneumothorax was made and the right lung failed to expand on repeated aspirations. Patient was unable to work. Weight loss of fifteen pounds.

Fig. 3, A and B.—A, Routine roentgenogram taken six months before onset of spontaneous pneumothorax, read as negative. B, Roentgenogram three months after occurrence of pneumothorax showing right lung 45 per cent collapsed. Fluid in left base. Extensive adhesions of right apex. Thickening of visceral pleural shadow.

Fig. 3, C.—Postoperative film showing expansion of right lung. Patient now well and working.

generally we do not have a valvelike mechanism, as there is usually a free communication with the bronchus. Thus, air is not trapped, and there is not as great tendency for these lesions to rupture as when a valvelike mechanism is present.

Four of the cases in our series showed evidence of pulmonary emphysema. Examples of this type of lesion are seen in Cases 6, 7, and 8. This finding was reported in 13 instances by Brock.¹² These cases are seen in an older age group. Our patients were 41, 44, 45, and 48 years of age. A history of previous repeated respiratory infection was given in all cases, which was not true of the second group. Some dyspnea on exertion had been present before the attack, but all of the patients were working and considered themselves only a little below par. In each of these cases large emphysematous bullae were seen, which were grossly similar to the more localized blebs. The intrapleural adhesions, seen in 3 of the cases near the bullae, may have exerted an abnormal tension on the bullae. Certainly, as will be pointed out later, they are an important factor in producing chronicity of the pneumothorax.



SPONTANEOUS PNEUMOTHORAX DUE TO RUPTURED APICAL BULLA

Fig. 3, D.—Drawing of right lung seen at operation. Right lung is 45 per cent collapsed and held captive by an inelastic visceral pleural membrane. Apical bullae are present with site of rupture at the base of one. Vascular adhesions hold lung at apex. Pleural fluid with fibrin clots is present. Resection of the bullae and decortication of the lung effected rapid pulmonary expansion.

CAUSES OF PERSISTENCE OF THE PNEUMOTHORAX

It is not always possible to tell exactly why the tiny rent in the wall of a bulla or cyst fails to heal, especially when there is complete collapse of the lung and mediastinal shift or herniation to the opposite side. Some of the openings are so small as to defy macroscopic identification without actually examining the

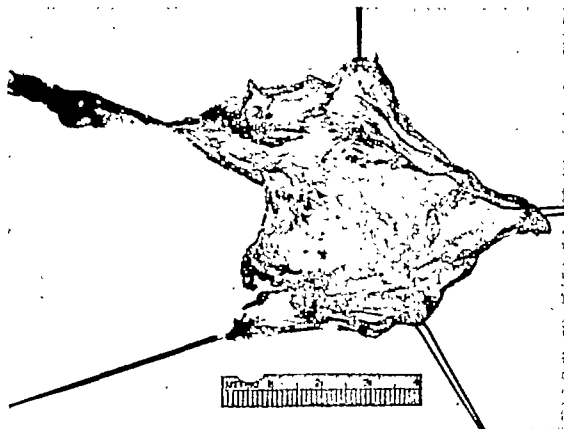
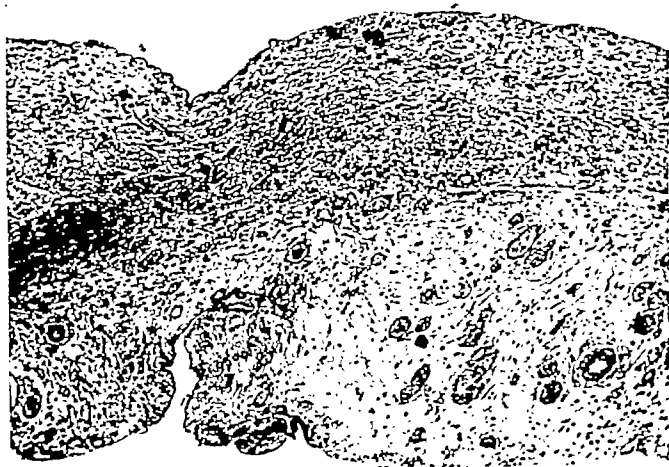
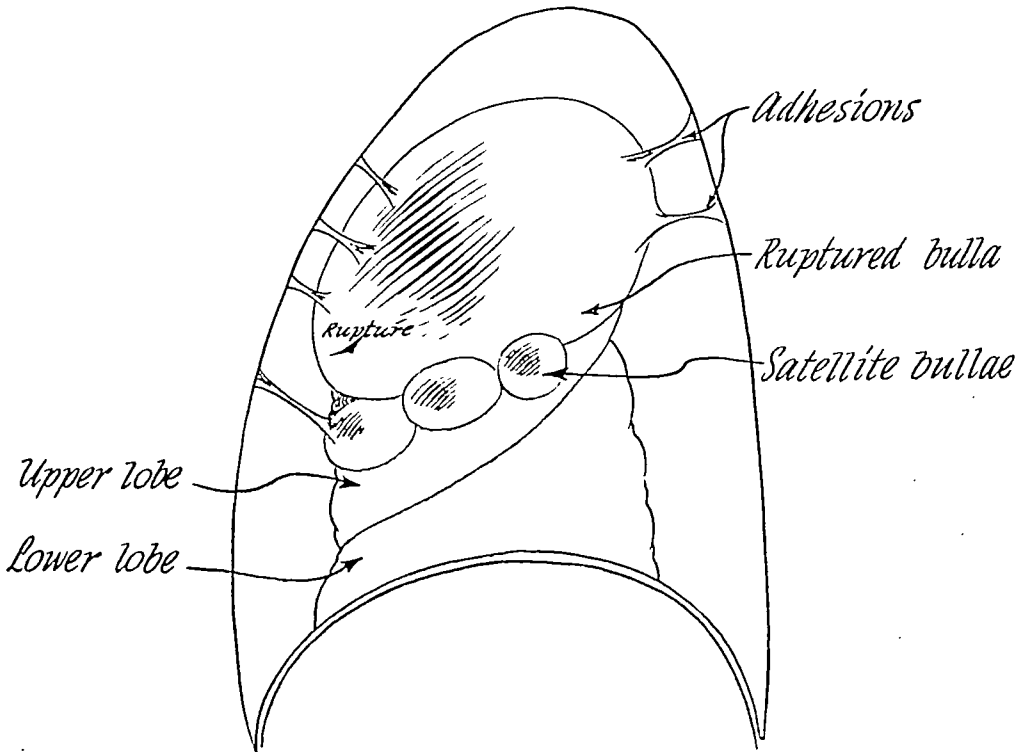
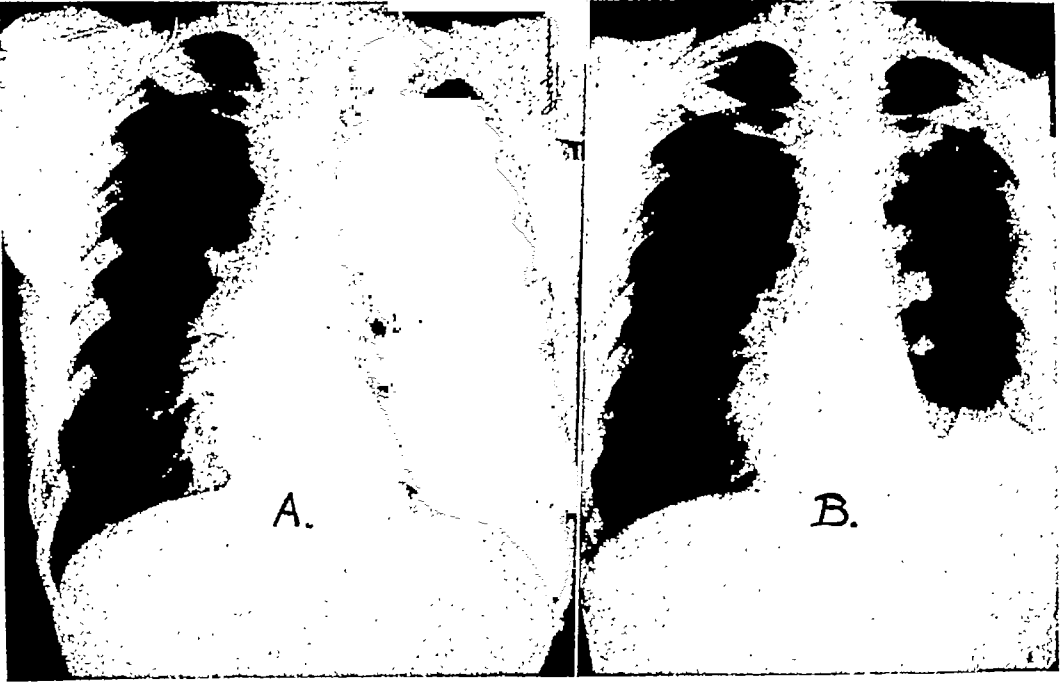
*E.**F.*

Fig. 3, *E*.—Gross specimen of part of fibroblastic membrane removed by decortication.

Fig. 3, *F*.—Photomicrograph of visceropleural membrane. The fibrous membrane is seen to be covered with mesothelium. A variety of inflammatory cells, foreign body giant cells, and capillaries is present.



SPONTANEOUS PNEUMOTHORAX DUE TO RUPTURED APICAL BULLA

C.

Fig. 4, A to C (Case 4).—A 36-year-old man was weak, tired, and short of breath following an attack of left chest pain three months previously. The attempts to expand the lung following the diagnosis of the pneumothorax had been unsuccessful. Patient had lost ten pounds, and had been confined to bed.

Fig. 4, A and B.—A, Roentgenogram three months after onset of pneumothorax. Left lung is 50 per cent collapsed. A suggestion of a large bulla is seen. B, Film six weeks after resection of the large bulla of left lung. The lung has completely expanded. Patient is now completely well.

Fig. 4, C.—Drawing of the findings at operation. Large apical ruptured bulla is held out by multiple adhesions. Several smaller bullae are present. Lung is 50 per cent collapsed. The lung expanded rapidly following pneumonolysis and resection of the bullae.

lung with the chest opened widely and testing for the aperture with saline solution and positive intrabronchial pressure. We recognize four factors as important in accounting for the failure of the lung to expand once an attack of spontaneous pneumothorax occurs: (1) intrapleural adhesions, (2) ruptured congenital cysts, (3) scar tissue in wall of bulla, bleb, or bronchiole, and (4) visceral pleural membrane.

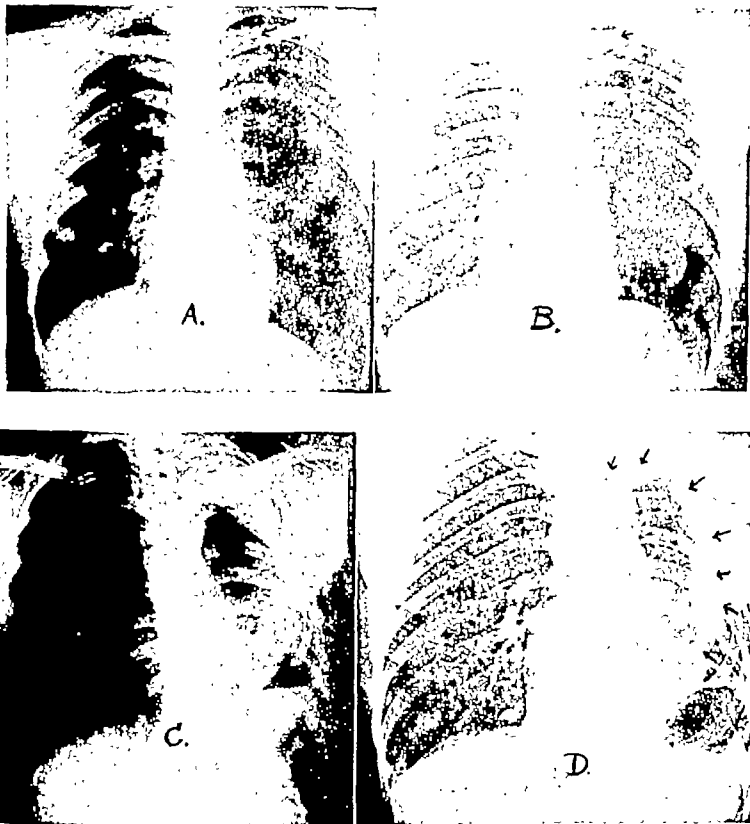
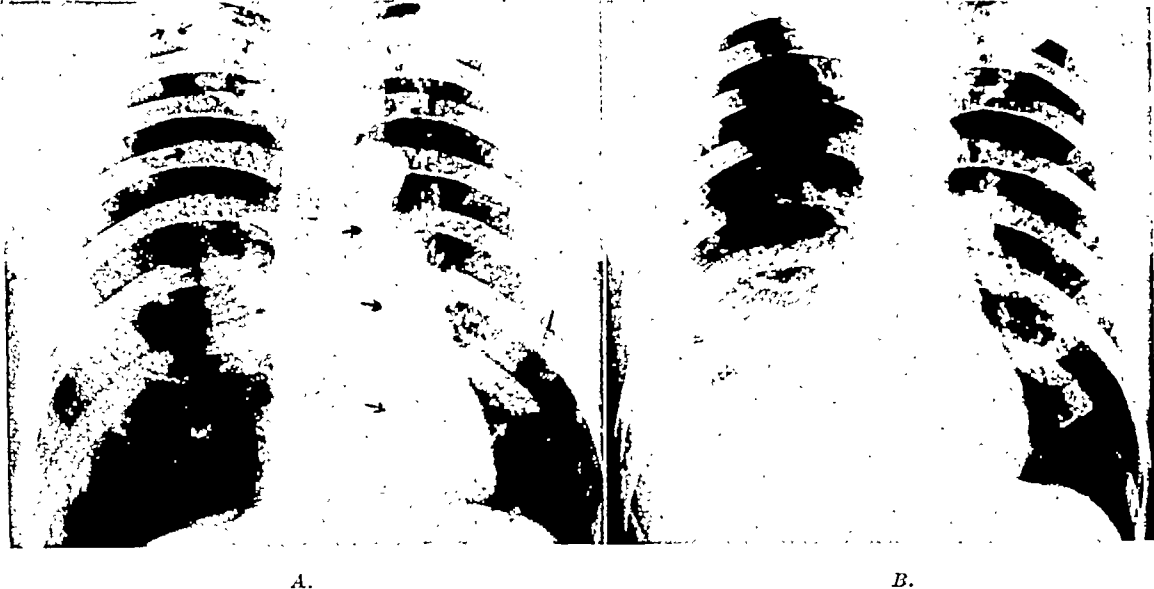


Fig. 5. A to D (Case 3).—A 37-year-old Japanese man had severe left chest pain and dyspnea for four months. Spontaneous pneumothorax was diagnosed and all measures to expand the lung had failed. Patient was a chronic invalid.

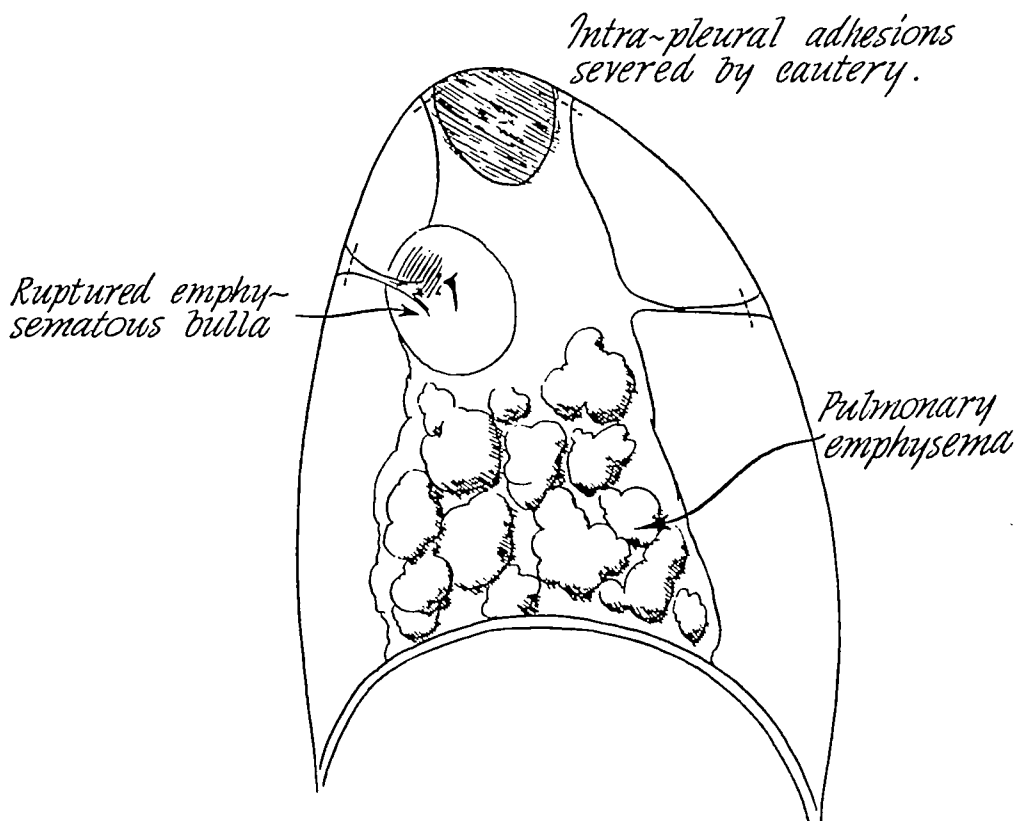
Fig. 5. A and B.—A, Pneumothorax left with 90 per cent collapse of lung, with intrapleural adhesions at apex of lung and diaphragm. Visceral pleura thickened. B, Following left phrenic paralysis no expansion of the lung. Thoracotomy then performed in 1911 with pneumonolysis and no decortication of lung, which we now believe was indicated in this case.

Fig. 5. C and D.—C, Two months after operation dense pleural shadow was present; no pneumothorax. Left diaphragm is high. D, Seven years later left pleurogenic fibrosis with shift of trachea and narrowing of intercostal spaces on the left. Left high frozen diaphragm. A decortication performed at time of pneumonolysis seven years previously would have prevented this.



A.

B.



SPONTANEOUS PNEUMOTHORAX DUE TO RUPTURED EMPHYSEMATOUS BULLA

C.

Fig. 6, A to C (Case 6).—A 41-year-old man complained of slight cough and expectoration for years. Following an attack of right-sided chest pain, which had been proved to be due to a spontaneous pneumothorax on the right, he had been incapacitated. Attempts to re-expand the lung were unsuccessful. The lung expanded following division of adhesions by internal pneumonolysis.

Fig. 6, A and B.—A, Right lung 70 per cent collapsed with apex of lung held out by fine stringlike adhesions. A mediastinal hernia is present. The heart is shifted to the left. B, Roentgenogram taken two months after the pneumonolysis. Right lung has expanded. Bilateral emphysema is present. Patient is now well and working.

Fig. 6, C.—Drawing of right lung as seen at thoracoscopy. Ruptured emphysematous bulla is seen at right apex. Lung 70 per cent collapsed. Multiple adhesions holding apex on tension were severed with cautery. Emphysematous changes are seen in the remainder of the lung.

Intrapleural adhesions were present in 10 of our 15 cases. These adhesions prevent the lung from collapsing completely, so that there is tension on the lung, and the pulmonary rent is held open. In some instances these adhesions may exert a "guy wire" effect, resulting in a valvelike action at the site of rupture of the lung. Since the air is trapped in the pleural cavity, positive pressure may be built up to produce mediastinal shift or herniation. In addition to the high percentage of occurrence of these adhesions in our series, there have been enough cases of spontaneous pneumothorax reported in the literature showing intrapleural adhesions to emphasize the importance of this type of lesion in maintaining the pneumothorax. Severing of the adhesions in four instances in our cases allowed the lung to drop away from the chest wall and the mediastinum. The rent sealed over, and re-expansion of the lung was rapid.

TABLE III. CAUSES OF PERSISTENCE OF CHRONIC SPONTANEOUS PNEUMOTHORAX
(AUTHORS' FIFTEEN CASES)

Pleural adhesions	10
Congenital cyst	3
Scar tissue in lung or bronchiole	5
Pleural membrane	7

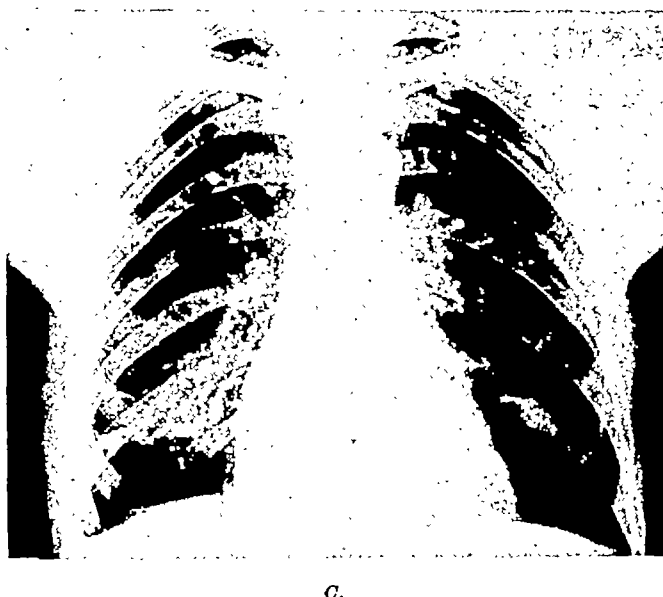
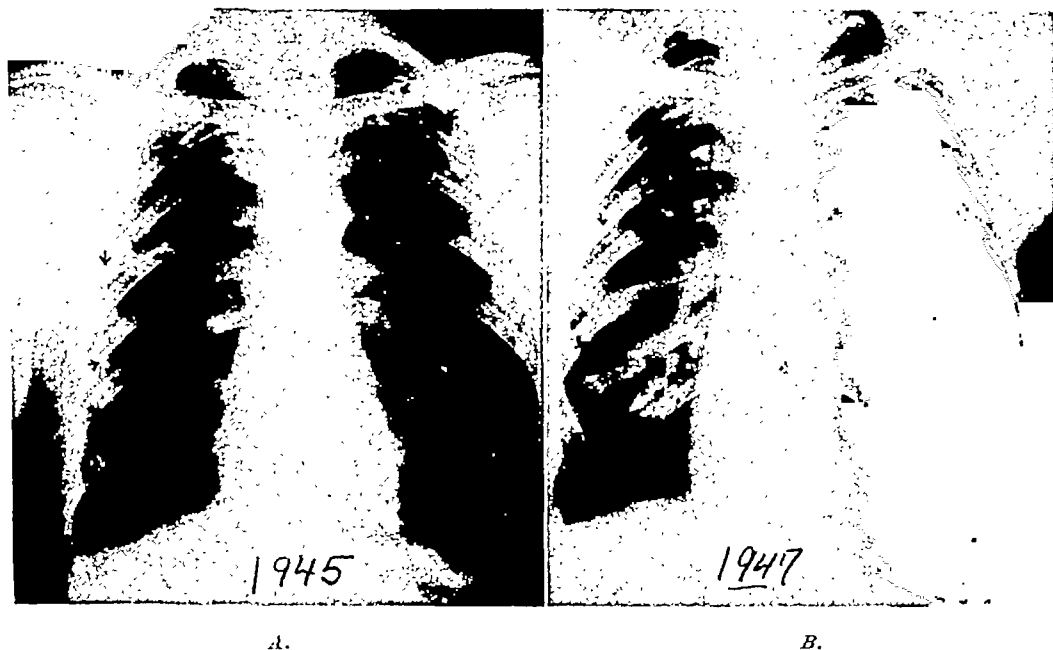
The structural characteristics of a congenital cyst are such that on rupture there is little tendency for spontaneous healing. The opening into the cyst is often a branch bronchus, lined by bronchial epithelium. The walls of the cyst are lined with cellular elements of the bronchial epithelium and bronchial walls. Since epithelial tissue does not seal over readily, the rapid healing of tears in the walls of a congenital cyst could not be expected. Thus, the very structure of congenital cysts tends to promote chronicity of the pneumothorax.

Localized blebs may be formed as the result of pulmonary and bronchiolar scars. Failure of these blebs to heal without the presence of intrapleural adhesions can perhaps be attributed to actual scar tissue in the lung or bronchiolar wall, which contracts to hold this aperture open. Yet the fact that the great majority of this type of bulla tend to heal spontaneously may mean that the valvelike mechanism and trapping of air does not persist after the lung is collapsed. When it does persist, a chronic pneumothorax may result. It is more difficult to understand why the rupture of these lesions results in chronic spontaneous pneumothorax than with the congenital type of cyst.

We have observed an additional phenomenon responsible for the persistence of the spontaneous pneumothorax, which has not been previously reported. A firm visceral pleural membrane was observed in 7 cases (6 of the 7 patients had surgery), to limit the expansion of the lung. This membrane may be formed on the visceral pleura as a result of the deposition of fibrin from the pleural effusion that occurs in some of these cases. When fibrin becomes organized, the lung is held captive in this tough envelope. In 1 case (Case 3) in which decortication was done, this membrane withstood a force of 1,500 Gm. without rupture. Fig. 3 *E* shows a photograph of a portion of the membrane which was decorticated from the collapsed lung in this case. Grossly it was

a tough, fibrous, gray-colored, opaque membrane, measuring from 1 to 3 mm. in thickness. Microscopic examination was reported* as follows:

Microscopic examination showed a membranous structure which consisted for the most part of fibrovascular tissue. The fibrous tissue varied from recently formed collagen to more compact scantily cellular collagen. The blood vessels



C.

Fig. 7, A to D (Case 7).—A 38-year-old man had repeated attacks of left chest pain and dyspnea diagnosed as spontaneous pneumothorax for two years. For three months patient totally incapacitated because of persistent collapse of left lung. Weight loss of fifteen pounds.

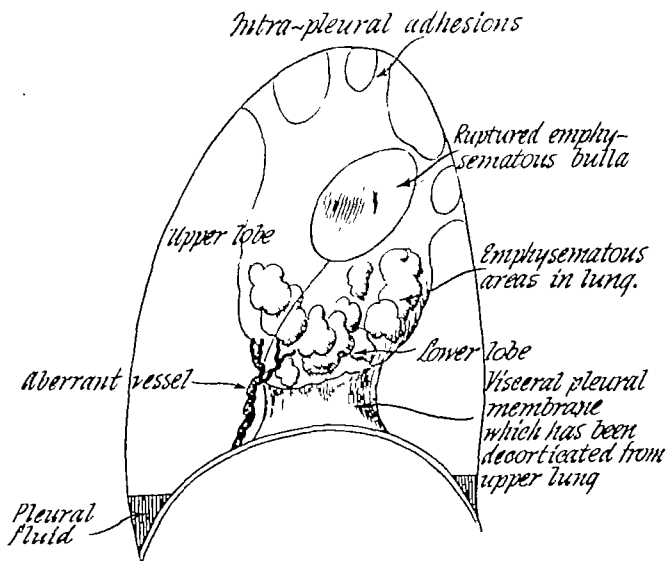
Fig. 7, A and B.—Chest film in 1945 shows small pneumothorax left with large emphysematous bullae on right; 1947, increase in the pneumothorax on the left.

Fig. 7, C.—Postoperative film shows complete expansion of left lung. Bilateral emphysematous changes. Patient now well and working.

*By Dr. L. S. Tragerman, pathologist.

were mostly capillary channels. A few arterioles were seen. A scanty leucocytic infiltration was noted. This consisted mostly of lymphocytes and tended to occur about vascular channels. Some portions of the surface of the membrane were covered by fibrin, some of which appeared old and hyalinizing. Such surface areas frequently showed mesothelial lining cells, at times with swollen cytoplasm and prominent nuclei. Multinucleated giant cells of the general appearance of the foreign body type giant cell were also seen scattered near the surface of such fibrinous zones. No tubercles were seen. There was evidence of focal hemorrhage interpreted as part of the recent surgical procedure. Fig. 3 *F* is a microphotograph of a portion of this membrane. We have encountered no mention of this factor in the medical literature. Since almost one-half of our cases showed this condition, it must be a fairly common cause of chronicity of the spontaneous pneumothorax.

Before the war we were inclined to regard all deposits on the pleura as "thickened pleura," regardless of the etiology. As a result of the extensive wartime experience²⁷ with traumatic hemothorax, it has been learned that fibrin is deposited on the pleura in layers. After varying periods of time it becomes

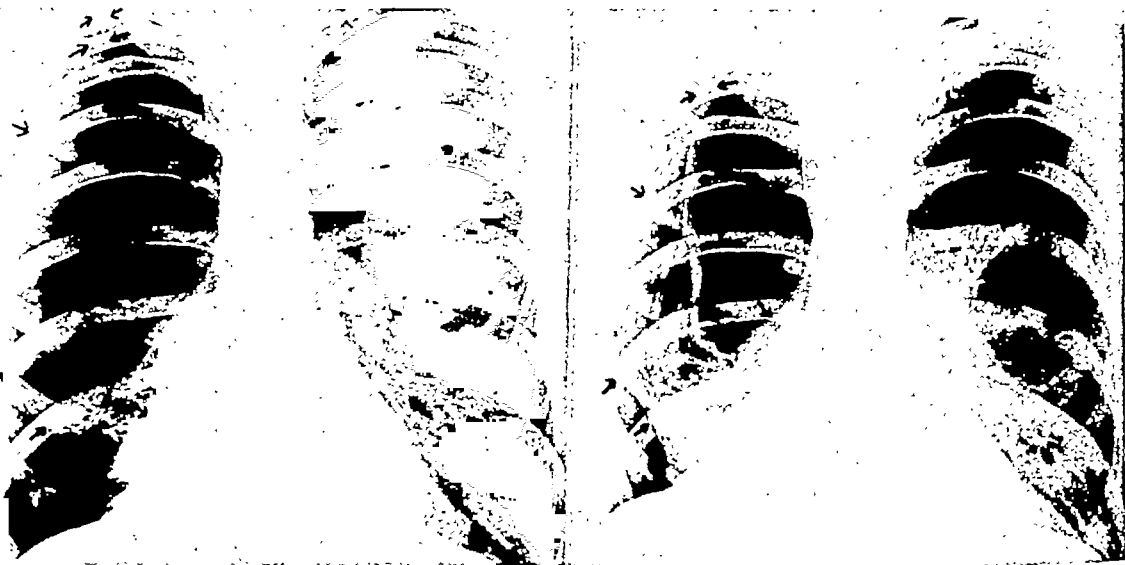


SPONTANEOUS PNEUMOTHORAX DUE TO
RUPTURED EMPHYSEMATOUS BULLA

D.

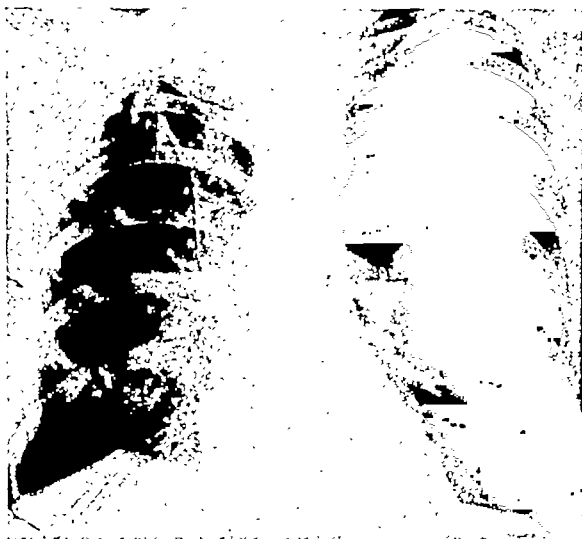
Fig 7, D.—Drawing of findings at operation. Ruptured emphysematous bulla is present in subapical region. Emphysematous changes are seen in the rest of the lung. Lung is 50 per cent collapsed and held by inelastic visceral pleural membrane. Pleural fluid is present. Anomalous vessel extends from diaphragm to lower anterior lung. Pneumonolysis, pulmonary decortication, and resection of bulla effected. The lung expanded rapidly.

organized to form a true fibroblastic membrane. The length of time that this membrane remains as a separate layer, which may be peeled off the visceral pleura, depends upon the presence of infection in the pleural cavity and the subpleural lesion in the underlying lung. As has been pointed out, because the rupture of the lung is on a mechanical basis and not due to acute pulmonary infection beneath the pleura, infection in the pleural cavity has not been noted



A.

B.



C.

Fig. 8, A to D (Case 8).—A 44-year-old man had chronic cough for years and mild dyspnea, but considered himself well. Three months before admission, following severe right-sided pain, patient had been diagnosed as having spontaneous pneumothorax. He was totally incapacitated and had lost twenty pounds.

Fig. 8, A and B.—A, Chest film showing pneumothorax right with complete collapse of right middle and lower lobes, and ill-defined huge cyst of upper lobe, held up by small adhesion. Generalized emphysema on left. B, Cyst of right upper lobe has collapsed more. Apical adhesion prominent. Thickening of visceral pleural shadow can be seen.

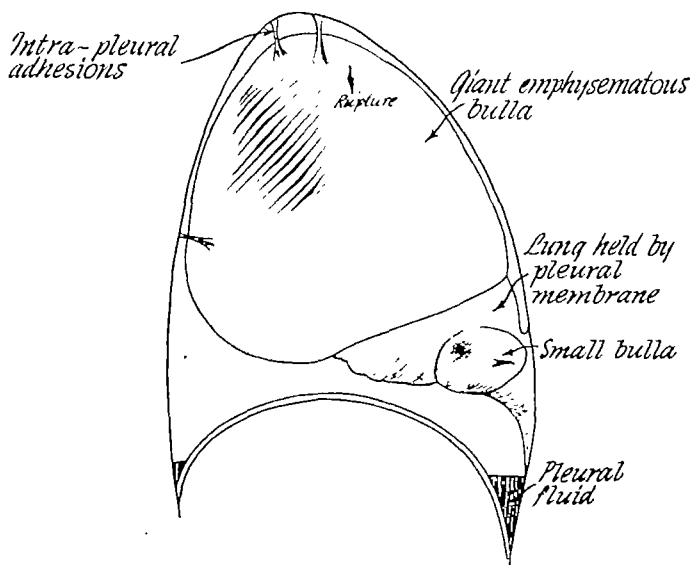
Fig. 8, C.—Postoperative film showing complete expansion of right lung. Bilateral emphysematous changes and fibrosis are present. Patient gained thirty pounds and is working steadily as an engineer.

in these cases. Thus the membrane does not become incorporated by fibroblastic proliferation as an integral part of the visceral pleura until several months have transpired, and it may be peeled off even after a period of five months.

DIAGNOSIS

The diagnosis of the acute attack of spontaneous pneumothorax is usually more difficult than that of the chronic state. The history of acute pain in chest or abdomen, dyspnea, prostration, and shock, coming on at rest or after moderate activity is well known. The clinical findings, leading to the performance of diagnostic roentgen examination, have been well emphasized in the literature¹⁵⁻¹⁸ and need no repetition here.

In the chronic state, usually the diagnosis has been established before the patient sees the surgeon. The patient complains of dyspnea on slight exertion, weakness, and ease of fatigue. The appetite is poor and the patients are usually underweight. A history of some pain in the affected side of the chest usually



SPONTANEOUS PNEUMOTHORAX DUE TO RUPTURED EMPHYSEMATOUS BULLAE

D.

Fig. 8. D.—Drawing of findings at operation. Small pneumothorax is present. A giant emphysematous bulla of the right upper lobe occupies most of the pleural cavity and is held out by intrapleural adhesions. Small point of rupture demonstrated near apex. Small satellite bulla is present on lower lobe. Pleural fluid present. Right lung markedly collapsed and held by inelastic pleural membrane. The two bullae were resected and the lung decorticated. Pulmonary expansion was rapid.

heralded the attack months before. On physical examination the patient may be either pale or of a dusky appearance, depending on the state of hypoxia that is present. Evidence of loss of weight is common, and breathlessness is seen with moderate exercise. The signs in the chest are those of fluid and air in the affected side and possible mediastinal shift. The affected side moves less and may actually be flattened. Râles indicating moisture in the bronchial tree or lungs are usually absent. The pulse is usually rapid after slight exercise, otherwise the cardiac findings are not noteworthy. We have not seen clubbing of the fingers in our cases.

On roentgen examination the pneumothorax is seen with varying amounts of fluid in the pleural cavity. In some instances the visceral pleural membrane shows up on the roentgenogram as a thickened pleural shadow. Usually it is impossible to detect the cause of the pneumothorax in the form of a bleb, bulla, or cyst of the collapsed lung by x-ray examination. The evidence of subpleural blebs or bullous emphysema on the affected or contralateral lung may be present. If a marked mediastinal shift is present, the contralateral lung may be compressed and show a mottled infiltration. In the differential diagnosis, it is important to rule out the presence of a so-called symptomatic pneumothorax. In most cases of the latter group the overt manifestations of the underlying disease are apparent. Careful history will usually elicit the symptoms of pulmonary suppuration or tumor, etc. Examination of the pleural fluid by smear and culture, and studies for tumor cells are indicated. Giant cysts of the lung may simulate a spontaneous pneumothorax. Evidence of trabeculae in the cysts or loculations of the cysts are important diagnostic aids. In some instances it is impossible to distinguish between a giant cyst and a spontaneous pneumothorax. We feel that in both instances surgical treatment is indicated. In one of our cases a giant congenital cyst held out by adhesions was present in addition to the pneumothorax (Case 2). In a second case a giant emphysematous bulla presented a similar finding (Case 8). Intrapleural pressure readings and the withdrawal of air in order to effect the expansion of the lung are indicated. However, one must proceed cautiously if a giant lung cyst is a possibility, particularly if the lesion is bilateral. The danger of puncturing a giant cyst wall and thus producing a pneumothorax is always present. We have done bronchoscopy in a few of these patients in which a bronchial block has been suspected or there was a productive cough. Bronchograms are indicated only when bronchiectasis is considered as being of clinical importance. All patients raising sputum should have a careful examination for tubercle bacilli by concentration and culture. In fact, the presence of cough and expectoration should warn the physician of a possible other diagnosis, or that he is dealing with a symptomatic pneumothorax. However, one of our patients with bullous emphysema did have an associated chronic bronchitis with cough and expectoration. With experience in treating these cases the diagnosis is usually obvious after a careful examination.

INDICATION FOR ACTIVE TREATMENT

We believe strongly that if the lung remains collapsed for a period of three months after an attack of spontaneous pneumothorax there is a definite indica-

tion for the institution of active therapy. The chances are exceedingly small that the lung will expand spontaneously after this period of time. In the past the inadequate treatment has been due partly to a lack of understanding of the factors responsible for the production and persistence of the pneumothorax. Failure to institute active treatment also stems from a belief that no active treatment is possible. Bed rest is important in the treatment of the acute spontaneous pneumothorax, but is entirely ineffectual in the chronic cases. The vogue for bed rest has been dictated by the idea that the lung will eventually expand and because the diagnosis of pulmonary or pleural tuberculosis has been made. As we have shown, both of these ideas are not well founded. However, the persistent pneumothorax may be just as disabling as pulmonary tuberculosis. Since the chest roentgenogram most often fails to demonstrate the presence of the etiological lesion in the expanded lung, we cannot be sure that a similar condition exists on the contralateral lung. Cases of bilateral spontaneous pneumothorax are being reported in the literature in increasing frequency. One of our patients, aged 23 years, developed a spontaneous pneumothorax of the left side following the successful resection of four pulmonary blebs for a spontaneous pneumothorax on the right. The fact that the right lung was successfully expanded following three years' pneumothorax, with the function of this lung preserved, certainly saved his life when the left lung suddenly collapsed. X-ray films failed to show any pathologic change in the left lung prior to the occurrence of the pneumothorax. The occurrence of a pneumonia in the uncollapsed lung may result in fatal outcome, as reported by Fisher.²⁸ We were called upon to see a patient in whom a chronic spontaneous pneumothorax had been present persistently for six years. Although a severe pleural reaction was present, the lung failed to expand. Because of dyspnea and chest pain, the patient was bedridden and had become a morphine addict. Attempts at expanding the lung were unsuccessful. He died of a coronary occlusion six years after the original attack of spontaneous pneumothorax. The impaired respiratory function and the hopeless outlook of the patient over a six-year period contributed to the coronary attack.

CHOICE OF TREATMENT

The two main forms of treatment have been employed to relieve chronic spontaneous pneumothorax: (1) artificial pleuritis by means of irritants introduced into the pleural cavity, and (2) surgical procedures.

Various types of irritants have been placed into the pleural cavity to set up a violent pleuritis, so that with the resultant pleural effusion and fibrin deposition the aperture in the visceral pleura and lung may be closed. A wide variety of pleural irritants have been used. These include Gomenol,²⁰ guaiacol,²⁰ iodoform,²⁰ iodized oil,²¹ dextrose solutions,²² silver nitrate,¹² iodized talc,²³ and others. The mode of employment and results have been well summarized by Brock,¹² and Hennell and Steinberg.²¹ The number of patients treated by the production of a sterile pleuritis is now well over 100, while in only 31 (16 in the literature and 15 in our series) has surgical treatment been applied. The strongest argument for the use of pleural irritants may be found in the fact

that Brock,¹² who has reported the largest series in the literature, chose this means of treatment for all but 10 of his patients.

The popularity of the use of irritants to cause a violent pleuritis is open to considerable question. Having been through the early days of "poudrage," as a preliminary operation prior to lobectomy, we have had considerable experience with chemical pleuritis. Poudrage was the technique of Bethune, whereby iodized talc was insufflated into the pleural cavity to produce a sterile pleuritis. The adhesions thus formed over the upper lobe tended to prevent the collapse of this lobe, should a bronchial fistula develop in the lower lobe. The pleuritis so produced resulted in such a violent constitutional reaction that the patients were often sicker following this procedure than after the lobectomy. This procedure has been entirely abandoned as preliminary operation to lobectomy. The postoperative course of our patients following thoracotomy has been mild, and the average postoperative stay in the hospital has been one week. The postoperative morbidity compares very favorably with the chemical pleuritis where weeks and months may be required to expand the lung.

It has been repeatedly shown by bronchspirometry tests that pleural effusion resulting in chronic pleuritis with fibrosis markedly decreases pulmonary function. In these cases the main indication of treatment is to preserve the pulmonary function at the highest possible level because of the danger of bilateral disease. With surgical treatment, rapid expansion of the lung is accomplished with minimal pleuritis, because the mechanical defect has been corrected. Minimal pleuritis means maximum pulmonary function. Thus, surgery is preferred to preserve pulmonary function.

In 7 out of 16 cases (15 surgical, 1 nonsurgical) we have observed that a definite pleural membrane was present as the result of a pleuritis in which there was a persistence of the pneumothorax. In these cases the production of additional pleuritis and pleural thickening would seem to be useless. The lung has expanded promptly following operation, and no prolonged period of hospitalization is necessary. Since the lesion responsible for the pneumothorax presents a mechanical problem, correction of this mechanical defect is logically a matter of surgery. When dense adhesions hold the lung aperture open, the deposit of fibrin in the pleura might serve only to produce a limiting membrane preventing re-expansion. Examples of this result have been reported.³¹ In spontaneous pneumothorax from congenital cysts or bullous emphysema, pleural irritants are not advisable. The subacute stage of a spontaneous pneumothorax produced by the rupture of small localized blebs would seem to be the most favorable indication for the use of these irritants. Then only those producing a mild pleural reaction should be used.

SURGICAL TREATMENT

The management of the acute form of spontaneous pneumothorax has been well covered in the medical literature^{15, 16} and consists of rest and the adjustment of the pleural pressures to permit a gradual expansion of the lung. The surgical measures for the treatment of chronic spontaneous pneumothorax in-

clude: (1) closed catheter drainage, (2) phrenic paralysis, (3) thoracoscopy with internal pneumonolysis, and (4) thoracotomy with the employment of various surgical procedures.

Closed catheter drainage is a measure which is efficacious mainly for the acute and subacute forms of spontaneous pneumothorax. It is important that the catheter be introduced in the second intercostal space anteriorly so that fluid or the fibrin will not clog the tube. We include this method of treatment for the sake of completeness. It might be tried in certain poor-risk chronic cases in the absence of demonstrable adhesions, congenital cysts, pleural membrane, or where tension pneumothorax might develop.

Phrenic paralysis was first employed by Weiss¹ in 1930 to relax the diaphragm and decrease the tension on the lung in order to permit the pulmonary aperture to heal. In 2 cases in the literature this means was successful.^{1, 2} We have attempted it in 1 case (Case 5, Fig. 5 *A* to *D*) with an unsuccessful result, because of the presence of mediastinal adhesions. We believe that phrenic paralysis is less desirable than more direct methods of treatment.

TABLE IV. TYPES OF SURGERY EMPLOYED
(AUTHORS' FIFTEEN CASES)

	CASES	SUCCESSFUL	FAILURE
Phrenic operation	1		1
Closed pneumonolysis	4	2	2
Thoracotomy:	13	13*	
With pneumonolysis	10		
With resection of cyst	10		
With decortication	5		

*One case lung expanded, but failure to do decortication resulted in marked pleurogenic fibrosis of lung.

Thoracoscopy is indicated as a diagnostic procedure and, when stringlike adhesions are present, as a therapeutic measure as well. Division of these adhesions with the cautery introduced through a thoracoscope was first successfully performed by Kipfer³ in 1932, in order to relax the tension on the lung to permit rapid healing of the pulmonary rent. After 3 pneumonolyses, the lung expanded. We did thoracoscopy in 4 of our patients. In 2 instances we were able to divide cordlike adhesions with resultant successful expansion of the lung. In the other 2 cases the adhesions were too dense for division. Case 6 (Fig. 6 *A*, *B*, and *C*) shows the type of adhesions that may be divided by internal pneumonolysis. Brock thorascoped all of the patients in his large series, mainly for diagnostic reasons, but also as a means of producing artificial pleurodesis, by means of the introduction of silver nitrate. In 2 cases adhesions were successfully divided. Open thoracotomy offers a better visualization of the lesion, particularly when the exact location of the pulmonary aperture is not certain, unless the lung is covered with fluid and positive pressure administered. At one sitting the definitive surgical treatment can be employed with the greatest conservation of pulmonary function. For this reason we have preferred thoracotomy to thoracoscopy.

Formerly we reserved thoracotomy for those cases in which phrenic paralysis or closed pneumonolysis failed. Now we believe that it is indicated as a

primary procedure. It has been used successfully by us in 13 instances to effect a cure. The senior member of our group (F.S.D.) apparently was the first one to perform a thoracotomy to relieve a chronic pneumothorax in 1929. The pneumothorax occurred in a 3-month-old infant as the result of a lung perforation in a congenital cyst following the introduction of an aspiration needle. Complete collapse of the lung occurred and persisted for three months. At operation the tear in the lung was found and sutured. A tubular adhesion, still dangling from the chest wall, was excised. The baby made an uneventful recovery. From this early experience we have evolved the present-day techniques. The surgical procedures available with the chest open are: (1) a complete pneumonolysis, (2) surgical closure of the pulmonary opening, (3) resection of the pulmonary cyst or bulla, and (4) decortication of the lung.

A complete pneumonolysis may be safely performed through the open thorax without fear of hemorrhage. The lung is completely freed of tension and small lesions will close spontaneously. Pneumonolysis was performed in 10 cases, in 2 of which it was the sole intrathoracic surgical procedure necessary. In these 2 cases the aperture in the lung closed as soon as the tension was taken off the lung. In the other 8 cases, additional procedures were performed.

Surgical closure of the ruptured bleb or bullae by suturing has been performed in 2 instances.⁹ We have not tried this as we believe resection of the affected area more effective and safe. In 1 of the cases in the literature⁹ death occurred on the first postoperative day, either of mediastinal emphysema or a cerebral embolus.

Surgical resection of congenital cysts and large localized blebs and emphysematous bullae is the most direct approach to this problem. First reported by Sycamore in 1936,^{3a} it has been performed 11 times in our series and 14 (5 local resections and 9 lobectomies) in the literature. Because we strongly believe that pulmonary tissue should be conserved, only the local or segmental resection of the pulmonary tissue should be employed. We have performed local resection in 10 cases, which included several extremely large cysts, and have not employed lobectomy in a single case. Lobectomy is to be avoided if possible, for the salvaging of even a few segments of a lobe may be important to the patient, if a contralateral pneumothorax occurs. Of the 9 cases of lobectomy reported in the literature (Brock, 3, and Hoyer and Clagett, 1), 1 patient succumbed to a contralateral spontaneous pneumothorax three months after operation. Examples of resection of the various types of cysts and bullae are reported in Cases 1, 2, 3, 4, 7, and 8.

As the result of extensive experience in World War II with the decortication of organized and infected hemothorax,²⁷ considerable speculation was voiced as to what the peace-time application of these lessons would be. In studying these cases of chronic spontaneous pneumothorax in light of our experience with battle casualties, we realized for the first time that the collapsed lung in these cases was held captive by a thickened fibroblastic pleural membrane in a manner similar to that observed in battle casualties with massive organized hemothorax. In fact, where such a pleural membrane exists, chemical pleurodesis and the surgical measures outlined previously will not be effective unless

this membrane can be removed. The techniques of decortication, standardized during World War II, are applicable here. We have found pleural membrane in 6 cases in this series.

We are surprised that this pathologic condition has not been described before, as there is no mention of this in the medical literature. In fact, an easily recognized pleural membrane (1 to 4 mm.), has been found in these cases. In one instance a tough, inelastic membrane withstood 1,500 Gm. of pressure without rupture (Case 3). The probable reason that this pleural membrane has not been described before is that only a few thoracotomies have been performed where the diagnosis could be accurately made. This condition may be only suspected by x-ray examination, and is easily overlooked on thoracoscopy. Actually we failed to appreciate the significance of such a membrane in the performance of an open thoracotomy for spontaneous pneumothorax in 1941, and failed to do decortication, which was definitely indicated (Case 5). Although the pleural opening sealed over, there developed a pleurogenic fibrosis with retraction of the chest wall, fixation of the diaphragm, and herniation of the upper mediastinum to the affected side. The effective pulmonary function of this lung must be very small indeed. Our experience with the rapid expansion of the lung following decortication in the 5 cases in which this procedure was done, and the minimal pleural thickening that follows this operation, leads us to believe that in Case 5 there would have been a great increase in the pulmonary function of the affected lung had we performed the decortication in 1941. Cases 1, 3, 7, and 8 present examples of combined decortication operation with resection of the cyst or bulla.

SURGICAL TECHNIQUES

Preoperative Preparation.—In preparing these patients for operation we have used the usual measures that are now commonly employed for pulmonary resection. In one instance where the pneumothorax was due to rupture of an emphysematous bulla (Case 8) the presence of a chronic productive cough necessitated several weeks' preparation with penicillin by intramuscular injection and aerosol route. Therapeutic bronchoscopy was helpful in improving the bronchial drainage (Case 8). In most instances the penicillin was administered only for several days before surgery and continued for two to three weeks after operation. Preoperative secondary anemia was amended by blood transfusion. Vitamin C and the B complex were administered in liberal quantities before and after operation.

Operative Technique.—The posterolateral approach was employed for the thoracotomy, using intratracheal anesthesia. In the younger individuals an intercostal incision was employed, while in older persons with a more rigid rib cage one rib was resected. After opening the pleural cavity, sterile saline solution was introduced into the pleural cavity to determine the source of the pulmonary rupture, much as one would examine a bicycle tire for a leak. With positive intrabronchial pressure administered by the anesthetist in all cases, except one in which we had freed the lung first, the site of the fistula was instantly obvious. Complete pneumonolysis was then done and the extent of the resection decided upon.

We have used simple resection of these peripheral lesions, clamping the lung and closing off the pulmonary tissue with mattress sutures of fine silk. In the case of large thin-walled cysts or bullae, it is well to open the cyst widely and proceed with the resection by keeping tension on the cyst wall. In this manner lung tissue may be conserved by the accurate identification of the attachment of the base of the cyst to the lung. It has not been necessary to do a segmental resection, though this procedure may be indicated, where the bleb lies close to the large pulmonary vessels. Actually we have dissected out the large pulmonary

vessels close to the bullae if we felt there was danger of catching the wall of these vessels with our suture. We have limited our resection to the lesions responsible for the pneumothorax and other peripheral large cysts or bullae which we felt might rupture at some future date. An attempt to remove all blebs or bullae is often technically impossible without pneumonectomy or lobectomy. We feel strongly that it is not only unnecessary, but is contraindicated if we are to conserve pulmonary tissue.

Decortication is performed if necessary, according to the standard techniques for clotted hemothorax.²⁷ We have found that the membrane peels off very easily. There has been very little oozing from the visceral pleura, which is shiny and glistening following the removal of the peel. The lung is carefully checked for bronchial fistula before closure. Tiny parenchymal bubbling, coming from the decorticated normal lung, is not serious and will be taken care of by the drainage tubes. Drainage tubes are inserted in the second or third intercostal spaces anteriorly and the sixth or seventh posteriorly, and are then connected immediately to a closed drainage system. The lung usually expands almost completely before the chest wall closure.

Postoperative Care.—The postoperative care is similar to that of a lobectomy, only the course is milder and the patients are not as sick. In most instances the patient feels the increase in breathing capacity as soon as he wakes up, and exclaims that he is using this lung for the first time in months. In only one instance was it necessary to aspirate tracheal secretions following operation. This was the case of a hypersensitive woman who refused to cough. Penicillin, in 100,000 units, is injected into the anterior drainage tube daily for two to three days, and the posterior tube is clamped for six hours after each injection. The drainage tubes are removed on the third or fourth days, as the lung has expanded and they cease to function.

COMPLICATIONS

There have been no complications in any of the adult patients incident to the surgical treatment employed. In a 3-week-old baby, the youngest patient in this series, a momentary cardiac standstill occurred during operation. This baby had spontaneous rupture of a congenital cyst soon after birth. In spite of closed catheter decompression of the pleural cavity, she remained in a precarious condition due to the large bronchial opening to the cyst. When we first saw her she was dehydrated, anemic, and in respiratory distress. After twenty-four hours' preparation we realized that the large fistula had to be closed if the baby was going to survive. The anesthesia was extremely difficult due to the baby's poor condition. A transitory cardiac arrest occurred, which responded to massage of the heart and the injection of 1/200 gr. of atropine sulfate. Following the operation she showed signs of cerebral malacia due to anoxia. These cleared in six months' time.

RESULTS

In all our series of patients there have been no recurrences and no deaths. All of the patients are leading fairly normal lives, as maximum pulmonary function has been preserved, with the exception of one (Case 5), where we failed to do a decortication. One patient, in whom a bulla was locally resected after collapse of this lung for almost three years, successfully withstood an attack of contralateral spontaneous pneumothorax, because most of the residual lung on the affected side had been preserved, and there was little pleuritis on the operated side. A number of the patients reported in our series have been operated upon during the past year, so we do not have long-time follow-up studies on these cases.

SUMMARY AND CONCLUSIONS

Chronic spontaneous pneumothorax is a potentially serious condition, as it leads to chronic invalidism which may be just as disabling as chronic pulmonary tuberculosis. Spontaneous pneumothorax is not "idiopathic" for, in the great majority of cases, the cause of the pneumothorax may be found in a ruptured subpleural cyst, bleb, or bulla. The etiology of these lesions is discussed. We recognize the important factors in the persistence of the pneumothorax as being the epithelial lining of congenital cysts, the presence of intrapleural adhesions, the scar tissue in the lung or bronchiole, and the formation of an inelastic visceropleural membrane. Since the pneumothorax persists because of mechanical conditions, surgery is indicated for their relief. The only other therapy has been the production of a pleurodesis by violent pleural irritant. We believe this method of therapy is definitely inferior to the surgical treatment presented, because of the limitation in pulmonary function so produced. Maximum pulmonary function should be preserved because of danger of a contralateral pneumothorax or other acute disease. The presence of the definite inelastic, fibroplastic, visceropleural membrane covering the lung is reported here for the first time in these cases, and the surgical importance of this lesion has been stressed. The technique of decortication of this membrane combined with pneumonolysis of the lung and resection of the ruptured pulmonary bulla in a series of 15 cases has been presented. The ideal procedure for the treatment of spontaneous pneumothorax is the one which will not only bring about obliteration of the pneumothorax pocket but also will return the best function of the lung. We believe that the surgical procedures outlined here will fulfill this ideal.

REFERENCES

1. Weiss: Quoted by Goldschmidt.²
2. Goldschmidt, W.: Phrenicusansschaltung beim pathologischen Pneumothorax, Wein. klin. Wchnschr. 44: 1276, 1931.
3. Kipfer, R.: Kasuistischer Beitrag zur Pathogenese des Spontanpneumothorax und zu seiner Therapie mit Hilfe der Thoracoscopie und endopleuralen kaustik, Verhandl. d. deutsch. Gesellsch. f. inn. Med. 44: 193, 1932.
4. Sattler, A.: Zur Behandlung des Spontanpneumothorax mit besonderer Berücksichtigung der Thorakoskopie, Wien. Arch. f. inn. Med. 30: 77-90, 1937.
5. (a) Sycamore, L. K.: Recurrent Spontaneous Pneumothorax, Am. J. Roentgenol. 36: 844-848, 1936.
(b) Tyson, M. D., and Crandall, W. B.: The Surgical Treatment of Recurrent Idiopathic Spontaneous Pneumothorax, J. THORACIC SURG. 10: 566-571, 1941.
6. Mouitt, E. R., Smith, J. V., and Eloesser, L.: Treatment of Spontaneous Pneumothorax, Dis. Chest 13: 221-36, 1947.
7. Horsley, J. S., and Biggers, I. A.: Operative Surgery, St. Louis, 1937, The C. V. Mosby Company, p. 605.
8. Hoyer, L. P., and Clagett, J. J.: Treatment of a Case of Spontaneous Pneumothorax by Lobectomy, J. THORACIC SURG. 16: 418-421, 1946.
9. Nissen, R.: New Operative Technik for Spontaneous Pneumothorax, J. Internat. Coll. Surgeons 8: 421-423, 1945.
10. Churchill, E. D.: Personal communication. Quoted by Tyson and Crandall.^{5b}
11. Cohn, R., Heaton, L. D., and Davis, J. K.: Treatment of Recurring Spontaneous Pneumothorax: Report of 2 Cases, Stanford M. Bull. 7: 15-22, 1949.
12. Brock, R. C.: Recurrent and Chronic Spontaneous Pneumothorax, Thorax 3: 88-111, 1948.
13. Brewer, L. A., III: The General Principles of Treatment of Acute and Chronic Forms of Spontaneous Pneumothorax, Bull. Los Angeles Surg. Soc. Vol. I. (In press).
14. Itard, J. E. M. C.: Sur le pneumothorax, ou les congestions gazeuses qui se forment dans la poitrine, Paris An. 11, 1803.

15. Kjaergaard, H.: Spontaneous Pneumothorax in the Apparently Healthy, *Acta med. Scandinav. Supp.* 43: 1-159, 1932.
16. Perry, K. M. A.: On Spontaneous Pneumothorax, *Quart. J. Med.* 8: 1-21, 1939.
17. Schneider, L., and Reissman, I. I.: Idiopathic Spontaneous Pneumothorax: History of 100 Unselected Cases, *Radiology* 44: 485-488, 1945.
18. Leach, J. E.: Pneumothorax in Young Adult Males: Description Statistics on 126 Cases, *Arch. Int. Med.* 76: 264-268, 1945.
19. Blackford, S. D.: Spontaneous Pneumothorax in College Students, *J. A. M. A.* 113: 737-739, 1939.
20. West, S.: The Bradshawe Lecture on Pneumothorax, *Brit. M. J.* 2: 293-400, 1887.
21. Diez: Quoted by Zahn.²⁴
22. Hayashi, J.: Vebir totlichen Pneumothorax durch Infarkt und Emphysem, Frankfurt. *Ztschr. f. Path.* 16: 1, 1915.
23. Fischer, B.: Der gutartige Spontanpneumothorax durch Ruptur von Spitzennarbenblasen, ein typisches Krankheitsbild, *Ztschr. f. klin. Med.* 95: 1, 1922.
24. Zahn, W.: Vebir die Entstehungsweise von Pneumothorax durch Constituistaistrennung der Lunguspleura ohne eitrige Eintzundungs, *Virchows Arch. f. path. Anat.* 123: 197-220, 1891.
25. Laennec, R. T. H.: *Traité de l'auscultation médiante*, II ed., Bruxelles, 1828.
26. Mayer, E., and Rappaport, I.: Pulmonary Emphysema: Newer Concepts, Friedman Lecture, National Jewish Hospital, Denver, Colo. 1944.
27. Sampson, P. C., Burford, T. H., Brewer, L. A., III, and Burbank, B.: Management of War Wounds of the Chest in a Base Center, *J. THORACIC SURG.* 15: 1-30, 1946.
28. Fisher, R. L.: A Case of Non-Tuberculous Pneumothorax Proven by Autopsy, *J. Michigan M. Soc.* 38: 774-776, 1939.
29. Candler, F. G.: Valvular Pneumothorax Treated by Mechanical Valve and Obliterative Pleurisy, *Lancet* 2: 639-640, 1939.
30. Apostolides, C., and Halussarantos, D.: Pneumothorax spontané avec fistule-soupape et épanchement puriforme traité au moyen d'injections d'huile gâicoolée iodiformée, *Paris méd.* 1: 338, 1930.
31. Hennell, H., and Steinberg, M. F.: Tense Pneumothorax: Treatment of Chronic and Recurrent Forms by Induction of Chemical Pleuritis, *Arch. Int. Med.* 63: 648-663, 1939.
32. Spengler, L.: Einiges zur Pathogenese, Prognose und Therapie des Spontanen Pneumothorax, *Schweiz. med. Wchnschr.* 2: 1276-1278, 1931.
33. Medici, F. A.: Results of Pleurodesis by Bethune Method in Benign Spontaneous Pneumothorax, *An. Cated. de pat. y. clín. tuberc.* 6: 106-115, 1944.

DISCUSSION

DR. RICHARD H. MEADE, JR., Grand Rapids.—Ten patients with chronic spontaneous pneumothorax were treated during World War II at Kennedy General Hospital, and one in Chicago later. Our experience has been closely parallel to Dr. Brewer's. Of the 10 patients at Kennedy, 3 had bronchiogenic cysts, and 1 of such extensive nature that a partial lobectomy had to be done; this man had evidence of cysts in the opposite lung, and evidence of cysts in the opposite lung, and evidence of cysts in the opposite lung. The two other cysts were easily removed with cystostomy in the lower lobe at the upper edge. 6 patients we could not find a definite etiologic evidence of deformity of the renal pelvis. lung which were sutured, but the chief procedure necessity of doing a lobectomy. In re-expansion. I think this is the most important point; all 6 had small openings in the thorax is due to this enveloping membrane. Decortication of the lung to allow

I should like to show a slide which illustrates the real cause of chronic pneumothorax. In our experience it could not be removed spoken of. In our experience it could not be removed of chronic hemothorax or chronic empyema. The next occasion to handle—a man who had extensive pneumothorax, the peel Dr. Brewer has just operating I decided to get pleural readings and to test the lung as readily as in cases all I accomplished was the production of an extensive intercostal space the last case I had extensive pressure that with the needle in place, air escaped interval months. Before was operated upon and it was found that he had multiple cysts, continuous drainage; the entire lung. We thought he also had emphysematous cystoma. He had such was obviously unwise to do a pneumonectomy and all we did was to remove the cysts, involving enfolding of one of the cysts. Drainage was carried out and the cysts, involving water trap. site lung. It is a caution and should be done with a

(Slide.) This was taken next day but you can see the extensive emphysema which was present. Only one tube was placed after the operation. That night it was necessary to introduce another tube in order to keep ahead of the air escaping from the pleural cavity. The next day he was "out of the woods" and the lung had begun to re-expand.

Five days later the lung had completely re-expanded and the tubes had been removed. (Slide.) This was taken a good many months later; he has continued to be well. I am sure he may have the same thing happen on the opposite side and he has been warned not to engage in too strenuous exercise.

This last case I think demonstrates the great value of closed drainage, whether using a water trap or active suction. I believe in most cases of chronic pneumothorax, even if it is impossible to demonstrate a definite lesion, if the lung is freed from its encapsulating membrane, and if closed drainage is applied, that will be all that is necessary.

DR. JULIAN MOORE, Asheville.—I would like to call attention to the danger of a hemorrhage in spontaneous pneumothorax. Ten years ago I saw one such hemorrhage which was fatal. The patient had a spontaneous pneumothorax; I saw him about twenty-four hours later and the hemorrhage continued in spite of repeated aspirations of blood and auto-transfusions. He was operated upon; one of the branches of the pulmonary artery in the upper lobe had been torn. The patient did not recover.

I have since seen 2 patients with hemorrhage associated with spontaneous pneumothorax. One of those patients recovered but was left with a fibrothorax because he was treated by late aspirations and was not submitted to surgery. Dr. Murphy and Dr. Elroy reported last year 1 patient who was seen three months after hemorrhage, and in whom decortication was successfully carried out and a good result obtained. About 50 cases have been reported in the literature in which spontaneous pneumothorax was associated with hemorrhage. If hemorrhage does occur, there is then the problem of controlling a serious hemorrhage as well as treating the pneumothorax.

DR. JOHN ALEXANDER, Ann Arbor.—I am discussing this paper to stress Dr. Brewer's warning against the indiscriminate injection into the pneumothorax space of glucose or blood or other irritating solution in an effort to seal the bronchopleural fistula. I realize that such solutions sometimes work well but, if they should fail to seal the fistula and if the lung should fail to expand, a thick membrane is likely to have been produced on both the visceral and parietal pleurae. This may permanently prevent the expansion of the lung and might prevent a subsequent successful decortication operation.

In our experience we have only infrequently found a pleural scar in patients having uncomplicated chronic spontaneous pneumothorax. I recall several patients whose chests have been incised, and who had had no evidence of a pleural scar or membrane; during the months before operation was finally accepted as necessary, there had been no evidence of pleural fluid. At the time of operation one or several ruptured blebs were found; the only steps needed at operation were (1) local excision of that part of the cortex of the lung that contained the blebs and (2) a gentle stroking of the entire visceral and parietal pleurae with dry gauze to promote complete obliteration of the pleural cavity after the lung had expanded.

After a wedge-shaped resection of a portion of the cortex, the sutures used to close the gap in the lung are under slight tension and might tear the lung if the pneumothorax air were rapidly evacuated by suction. I prefer to leave the lung partially collapsed while firm healing of the sutured area is taking place; absorption or intermittent aspiration of the pneumothorax air gently and gradually expands the lung. I am not sure, however, that this method is preferable to immediate expansion by suction drainage.

Dr. Blades has had great success from the local application of talc powder to areas in which bronchopleural fistulas are seen at the time of a thoracotomy.

DR. ARTHUR S. W. TOUROFF, New York.—I think that all of us are agreed that if the lung is collapsed and bound down by a fibrotic membrane, surgical treatment, namely, decortication, is indicated. However, I would like to say a word in defense of conservative

treatment of chronic pneumothorax of the usual variety. In that connection, I refer you to two articles by Dr. Herman Hennell of our Medical Thoracic Group. One appeared in the *Archives of Internal Medicine* for April, 1939, and the other in the *Journal of the Mount Sinai Hospital* for September, 1946.

Patients suffering from chronic pneumothorax naturally come to our medical men first. Since the latter traditionally are more conservative than we of the surgical guild, they have treated such cases almost routinely by the use of pleural irritants. They employ a saturated glucose solution of approximately 67 per cent concentration; and, when the lung is approximately three-quarters expanded, they introduce 10 to 15 c.c. of Lipiodol. They have treated 26 such cases up to the present, and, thus far, have been so successful that we of the Surgical Group have not been called upon to render surgical treatment. Perhaps we will some time in the future but, up to the present, we have not.

Of the 26 patients treated conservatively by our Medical Group, 23 have been cured with one course of injections. A course consists of four to six injections. Two of the 26 were cured with two courses and follow-up study could not be done in one. In other words, of the 25 patients in whom follow-up has been done, all are well.

Two patients with large air cysts of the lung were treated by the same method. The surgeons among us were shocked to see the medical men treat cysts of the lung in this way, but much to our amazement, the 2 patients recovered.

(Slides.) With regard to the first case that Dr. Brewer showed, you may recall that after the patient had been treated by underwater drainage, the lung expanded well. I think that our medical men would treat such a case conservatively. (Slide.) This is the film of a patient who already has had 67 per cent glucose introduced into the pleura. You will note that the lower portion of the lung has re-expanded. There is a large pneumothorax above. The heart is markedly displaced toward the right. (Slide.) Here is the film of the same patient after the introduction of Lipiodol. I might add that when irritating substances are introduced into the pleura, the patient is instructed to lie on his side for approximately twenty-four hours with the well side down so that the solution may come in contact with the surface of the lung. (Slide.) Here is a film of the same patient subsequently, with the lung fully expanded. Here you see globules of Lipiodol in the pleura, most of it lying behind the heart. (Slide.)

Here is a final case in which you see the lung completely collapsed. (Slide.) This is the last film demonstrating complete re-expansion of the lung and scattered droplets of Lipiodol in the pleura.

DR. FRANCIS WOODS, Brookline, Mass.—I would like to mention a small group of cases we have seen that falls, I think, into the same category. These patients have had repeated spontaneous pneumothorax, as much as six and sometimes eight times, and I think in these cases we have justifiably done explorations on the chests and found a small cyst which was the source of the leak, ligated it or removed it by segmental resection. In one or two who had repeated spontaneous pneumothorax no leak was found. Therefore, I would like to call attention to a group who do not have chronic spontaneous pneumothorax, but recurrent pneumothorax that I think should be treated in the same group.

DR. FELIX A. HUGHES, Jr., Memphis.—I think the plan of treatment presented by Dr. Brewer is rational and effective. The morbidity is much less in these patients if an aggressive attitude is taken toward the spontaneous pneumothorax. I would like to mention that of 15 patients with an acute spontaneous pneumothorax, treated with immediate closed thoracotomy, 13 had immediate re-expansion; 2 failed to have expansion and open thoracotomy was done with conservative resection of the bleb or cyst. I think the rupture or disruption of the lung or bleb may heal more rapidly and more permanently with the lung expanded than with it collapsed.

I would also like to present one slide which shows the membrane Dr. Brewer spoke of; here you can see a chronic spontaneous pneumothorax with one of the blebs excised. You can see other blebs in place in the lung.

DR. SHELDON E. DOMM, Knoxville.—Dr. Brewer has mentioned the rarity of autopsy confirmation in these cases. We would like to present a case in which there were unusual post-mortem findings. (Slides.)

This is a 14-year-old white boy, first seen with what appeared to be a simple acute spontaneous pneumothorax on the right, which responded to conservative management, but recurred in one month. Again the right lung re-expanded and appeared normal. However, two months later x-ray examination showed shadows suggestive of bullous emphysema in the right lower lobe. About one month after this development the patient was readmitted to the hospital in his third episode of right-sided spontaneous pneumothorax. Conservative measures, including an indwelling cannula, failed to relieve the dyspnea; subcutaneous and mediastinal emphysema ensued and were not relieved by supraclavicular and suprasternal incisions. X-ray examination three days later, on the day of death, showed another spontaneous pneumothorax to have occurred on the left. The patient died about six months after the first episode of spontaneous pneumothorax. Autopsy showed both lungs to be almost completely replaced by cyst formation with fibrosis, with emphysematous blebs studding the entire pleural surfaces. The pancreas was about one-fourth normal size and showed marked alteration of the normal architecture, notably a diffuse increase in connective tissue. This picture, however, differed from the usual findings in fibrocystic disease of the pancreas in that the pulmonary changes were decidedly atypical.

This case was particularly interesting to us because of its diagnostic and post-mortem features, and the question whether surgical intervention would have been justified.

DR. ALFRED GOLDMAN, Los Angeles.—I wish to bring out two points which have not been mentioned, that were encountered in operating on 4 patients with chronic spontaneous pneumothorax associated with large cystic dilations which one would probably classify as giant bullae. The first point is that on making the incision to open the chest the patient's condition is usually not good from the anesthetist's point of view. The anesthetist complains of some difficulty with the anesthesia; since the cyst usually appears in the wound as soon as the pleura is opened one should immediately puncture the cyst. When this is done the anesthetist usually will say that the patient's condition is improving and anesthesia proceeds more smoothly. The second point I wish to make is regarding the demonstration, in one of these patients, of the leak in the lung. Dr. Brewer mentioned that one must use saline solution; however, sometimes the point of rupture is not obvious and in one of these patients a two inch cyst was completely enclosed within an apical adhesion so that all one saw on opening the chest was the collapsed lung and adhesion. This was a large vascular villike adhesion, and on re-expansion of the lung and making pressure with the bag by the anesthetist, one could see the balloonlike cyst structure inflating through the walls of the adhesion. On using saline solution and putting the patient in the Trendelenburg position, a small rupture was discovered at the base of the adhesion. Thus one should look for a cyst within an adhesion if adhesions are present and no other obvious source for the air leak is discovered.

DR. BRIAN B. BLADES, Washington.—Dr. Alexander has mentioned my interest in the procedure of poudrage. I want to make it clear that, in my opinion at least, poudrage has no place in treatment of chronic pneumothorax. It is useful, however, for the patient who has frequent bouts of spontaneous pneumothorax of nontuberculous origin, and in whom at operation many small blebs are found over the surfaces of the lung. In some cases there may be dozens of blebs, all of which are the potential source of an air leak. I feel very strongly that if the seriousness of pneumothorax justifies operation, it justifies open operation, enabling direct inspection of the lung. In many cases a single, two, or perhaps three blebs are seen which can be excised. The use of poudrage is rather limited and only applicable to those cases in which there are many small blebs.

DR. BREWER.—I wish to thank all the discussants for the great interest that they have shown in this discussion. I think Dr. Touroff has demonstrated very well the effect of pleural irritants. Certainly, the second case that he presented showed considerable pleural

thickening. I think the use of irritants may be justified very early in the course of the disease, when the lung is mobile. Then, only, the injection of mild irritants might be tried. All who have been through the poudrage technique, with iodized talc, developed by Norman Bethune, as a preliminary procedure to lobectomy, know that the violent pleuritis frequently seen was serious; the patient often had more reaction to the poudrage than to the lobectomy that followed. This procedure, as you know, is not commonly used any more. Of course, if there are bilaterally destroyed lungs, no treatment will avail. However, the local resection (and by local resection we mean only the bleb that is responsible for the pneumothorax or a large thin bleb that is adjacent) will result in closing of the pleural aperture with minimal loss of pulmonary tissue. Certainly, we have reached the stage in the management of thoracic diseases where we must think of preserving pulmonary function and not merely the palliative sealing off of the pleural aperture.

We admit that in many instances the production of a violent pleuritis will seal over the pleural opening. However, it may take as long as one year for this sealing off to take place. This result is effected at the expense of pulmonary function.

SEGMENTAL RESECTION IN PULMONARY DISEASES

J. MAXWELL CHAMBERLAIN, M.D.,* AND (BY INVITATION)
COMMANDER THOMAS C. RYAN, UNITED STATES NAVY†
NEW YORK, N. Y.

INTRODUCTION

SEGMENTAL resection of the lung is an operation designed to rid the patient of the major diseased components with the minimum sacrifice of normal functioning parenchyma. This result is achieved if (1) the proper segment or segments are removed; (2) the residual circulation is not impaired; and (3) re-expansion is complete and immediate.

REMOVAL OF THE DISEASED SEGMENT

The wrong segment was removed in two instances in this series as demonstrated by postoperative bronchograms. Other mistakes may have been made in the few nontuberculous patients who did not respond to the request for follow-up bronchographic studies. The known errors, however, occurred early in our experience and if the present clinical evidence is reliable the number of other errors are few.

Basic anatomic knowledge of the bronchopulmonary segments is essential to a successful result, but often disease so distorts the normal anatomy that the surgeon is provided a new surgical experience at each resection. These alterations by disease in addition to the large number of (anticipated) anatomic variations have taught us the necessity for a standardized operative approach.

The first objective has been the identification and isolation of the bronchus serving the diseased segment. In many instances it is possible to divide the proper bronchus before ligation of *any* pulmonary vessel. In exposing the bronchus and its main divisions it may be necessary to define several branches of the pulmonary artery to a lobe before the proper bronchus may be selected.

The second objective is ligation of the arterial supply to the segment. It is often technically much easier to divide the artery before the bronchus, but the inconsistency of the vascular pattern in comparison with that of the bronchial tree has influenced the order of our approach. Once the bronchus is divided and gentle traction is put upon it, the proper segmental artery quickly joins it. Only then is it safe to ligate and divide it.

The final objective in the removal of the bronchopulmonary segment is ligation of the pulmonary veins. Suffice it to say, at this time, that it is a good general rule to ligate as few veins as possible at the hilar level.

The opinions or assertions expressed in this paper are private ones of the authors and are not to be construed to be official or reflecting the views of the Navy Department or the Naval Service at large.

Read at the Twenty-ninth Annual Meeting of The American Association for Thoracic Surgery at New Orleans, La., March 29-31, 1949.

*Thoracic Surgeon, Pack Medical Group, New York, N. Y.

†Chief, Chest Surgical Service, St. Albans Naval Hospital, St. Albans, N. Y.

RESIDUAL CIRCULATION

The veins are intersegmental and receive tributaries from two adjacent segments. Ligation of the vein at the hilum may cause, thereby, a passive congestion of the segments to be left behind. Swan⁷ has recently reported the pathologic changes associated with ligation of the pulmonary veins. These changes are mainly those of passive congestion, and if after segmental resection postoperative hemoptysis or streaking occurs beyond the first few days, it is suggestive evidence that venous obstruction is present in the residual circulation.

The proper segmental plane for dissection is disclosed as soon as gentle traction is applied upon the divided segmental bronchus and artery. It is in this plane that the small venous tributaries empty into the main intersegmental vein. As the dissection progresses the larger tributaries may be clamped, but the smaller ones are usually torn across unseen at their junction with the intersegmental vein. A small amount of bleeding occurs, as a result, but this soon stops, and the intersegmental vein is easily visible on the raw surface of the residual segments. Preservation of this vein is an important refinement in the technique of segmental resection. This procedure, as outlined, is a true segmental resection.

RE-EXPANSION

Complete and immediate re-expansion of the lobe and segments is essential to a successful result because (1) infection may be prevented if the dead space is obliterated and (2) maximum function of the residual parenchyma is obtained.

Obliteration of all dead space is a basic principle in surgery if infection is to be avoided. It is an accepted fact that the pleural cavity is always contaminated in the resection of the lung, a lobe, or any of its segments, but it is also recognized that the pleura like the peritoneum is not entirely defenseless. Therefore, if one is careful to reduce the contamination to a minimum and is insistent upon complete and immediate re-expansion, an empyema is not likely to occur. This principle was true and effective before the use of chemotherapy, yet even with chemotherapy an empyema occasionally develops if this fundamental principle is violated.

Maximum respiratory function, of course, is the main objective in establishing complete and immediate re-expansion, but if this end is to be realized, constant and controlled intrapleural suction is considered essential. A few small fistulas usually are present after a segmental resection and in a sense the problem is similar to the one which occurs in a spontaneous pneumothorax. Its treatment, however, is quite different. The lung or segments should not be allowed to recoil until the fistulas have closed, because a serosanguineous effusion (simulating a hemothorax) accompanies the thoracotomy, and the lung may be trapped in the partially collapsed position. Continuous but controlled suction which can be adjusted to maintain the normal intrapleural pressure seems preferable and is, of course, more physiologic. Such suction removes air faster than it leaks through the fistulas and in this way a normal negative pressure is established in the pleural cavity.

Adjacent structures soon fuse with the raw area and seal the fistulus so that re-expansion is rapid and complete. For example, if the superior segment of the lower lobe has been preserved, the diaphragm soon rises to seal the raw surface; if the apical posterior segment of the left upper lobe has been removed, the raw area of the anterior segment is soon fused in the vertebral gutter; and in a similar way, in each segment removed, the diaphragm, the adjacent lobe, the chest wall, or the mediastinum soon fuse with the raw area and all fistulas are closed.

The actual contribution of the residual segment to total lung function is probably not great, but as a "filler" it contributes greatly to respiratory function. For example, in those cases in which the superior segment of the left lower lobe has been preserved, a large pyramidal space is filled by the segment. The diaphragm, as a result, not only retains its normal contour, but, more important, retains its pistonlike activity. On the other hand, if the entire lobe is removed the upper lobe must overdistend considerably to fill this space, and the diaphragm becomes elevated, flat, and fixed in the vertebral gutter. For obvious reasons, diaphragmatic fixation and overdistention of the lung are complications to be avoided whenever possible. Therefore, the main contribution of the residual segment is its influence upon the function of the diaphragm or the other components of the lung.

TECHNIQUE

The segmental resection technique which we have used was based upon the concepts of Churchill,³ Blades,¹ Clagett,⁴ and Overholt.⁵ We began as did Churchill by identifying, isolating, and dividing the cardinal structures (except the vein) at the hilar level. We applied this technique to individual segments as suggested by Blades. We used Clagett's "traction-dissection" technique, but without closing the raw area as advocated by Overholt.

However, early in our experience we discovered the unreliable nature of the venous pattern and errors were made by ligation of the wrong veins. This led to a state of insecurity on our part, the solution to which was procrastination. We decided to put off the ligation of any vein as long as possible. Therefore, after selecting the proper bronchus and artery, the intersegmental plane was entered and the dissection continued until no further progress could be made without ligation of a vein. Once the segment was removed, however, there could be seen a large vein on the raw surface of the adjacent segment which bled at many points along its course. Only then did we understand the teachings of Boyden and Seannell,² that there were intersegmental veins which received blood from two adjacent segments. Finally, we realized that during the dissection we were tearing across venous tributaries which were enroute to join the main intersegmental vein. Overholt referred to them as "vessels" which were often indistinguishable from the smaller bronchioles and he recommended severing them. If bleeding occurred they were ligated, if not, they were traced as small bronchi to their termination and removed with the segment. Though we have refined our techniques and now clamp and ligate the larger tributaries, we still find it difficult to avoid traumatizing the intersegmental vein in all cases. The

injury is often due to the fact that the inflammatory reaction, at one time, has extended across into the adjacent segment; the residuals of this process interfere with dissection along the intersegmental plane. Obviously, therefore, attempts at segmental resection during an acute inflammatory phase are to be avoided. In this connection, however, if during dissection the intersegmental plane seems to be obliterated by the chronic inflammatory reaction it may be advisable to return to the parent bronchus and select an adjacent bronchus which also is included in the resection. The entire inflammatory process is circumvented in this way. Occasionally, a subsegment of the adjacent segment will suffice. In other words, a segment and subsegmental resection are done.

Other techniques are indicated in some cases. For example, when the lesion is small the subsegment may be removed by local excision. A modification of this technique is the excision between clamps when the lesion is located along a fissure or the free edge of a lobe. Occasionally, it is preferable to begin the resection at the periphery. We have referred to this as the "outside-in" technique. Cysts, atelectatic segments, or even chronic lung abscesses may be treated in this way. Our experience would lead us to conclude, however, that those methods which leave the raw area exposed and do not alter the contour of the residual lobes for the reasons we have pointed out are preferable to those which prevent the lung from "breathing" by approximation of the pleural surfaces.

SUMMARY OF CLINICAL MATERIAL*

The conclusions on segmental resection are based on the experience gained in this series of 100 segmental resections. A total of 91 operations were performed on 85 patients in the last two years (Table I). This type of operation has been done on 37 patients with bronchiectasis, 30 with tuberculosis, 13 with lung abscess, 2 with cysts of the lung, and 1 case each of bronchiogenic carcinoma, bronchial adenoma, and coccidiomycosis.

TABLE I. RÉSUMÉ OF THE STATISTICS

	PATIENTS	OPERA- TIONS	SEGMENTS RESECTED	COMPLI- CATIONS	DEATHS
Bronchiectasis	37	43	52	19	1
Tuberculosis	30	30	30	10	1
Lung abscess	13	13	13	1	1
Other diseases:					
(1) Cysts	2	2	2	0	0
(2) Coccidiomycosis	1	1	1	0	0
(3) Bronchial adenoma	1	1	1	0	0
(4) Bronchiogenic carcinoma	1	1	1	0	0
Total	85	91	100	30	3

BRONCHIECTASIS

In the cases of bronchiectasis (Table II) there were a total of nineteen complications, but several of these complications occurred in the same patient so that the complications occurred in less than the 44 per cent which seems to

*The material for this paper came from the Chest Surgical Service of Bellevue, St. Albans, Halloran, and Triboro Hospitals. In eight of the cases from the Service at Bellevue in which this technique was used, the patients were not operated upon by the authors, but permission for their inclusion was granted by Dr. Berry, the Chief of the Thoracic Service at Bellevue.

TABLE II. SEGMENTAL RESECTION FOR BRONCHIECTASIS

PATIENTS	OPERATIONS	SEGMENTS RESECTED	COMPLICATIONS	DEATHS
37	43	52	19	1
Complications in the Bronchiectasis Cases				
	Spread, contralateral		1	(Patchy pneumonia)
	Bronchopleural fistula		3	
	Empyema without bronchopleural fistula		1	
	Incomplete or delayed re-expansion		5	
	Atelectasis		3	
	Recurrent spontaneous pneumothorax		3	
	Pulmonary embolus		1	

be indicated. The complications were not serious and added little to the dangers of the operation in most instances. Atelectasis required bronchoscopy in only one instance, while the other two patients responded rapidly to intratracheal aspirations with a catheter. The incomplete or delayed re-expansions required suction on intercostal tubes longer than the forty-eight to seventy-two hour period usually necessary. One patient required repeated aspirations over a period of several months. The cases of spontaneous (recurrent) pneumothorax required closed thoracotomy drainage by an intercostal tube connected to suction for only the forty-eight to seventy-two hour period. The three patients with bronchopleural fistula who developed localized empyema required intercostal tube drainage for a period of several weeks. The one case of empyema without bronchopleural fistula probably was due to breaking into a localized abscess surrounding a piece of shrapnel in the lung which was believed to be the cause of the bronchiectasis and was removed at the operation. The one mortality in this group was probably due to pulmonary embolus.

TUBERCULOSIS

In the thirty cases where segmental resection was done for tuberculosis (Table III), the early postoperative complications were not as severe nor as numerous as would be anticipated. There were three of these cases in which a lobectomy was performed on the same side at the same operation and one case in which segments were removed from two lobes at the same operation, so the complications must be considered in light of these compound procedures.

TABLE III. SEGMENTAL RESECTION FOR TUBERCULOSIS

PATIENTS	OPERATIONS	SEGMENTS RESECTED	COMPLICATIONS	DEATHS
30	30	30	10	1
Complications in the Tuberculosis Cases				
	Spreads, contralateral (or reactivation)		1	
	Spreads, homolateral (or reactivation)		2	
	Bronchopleural fistula		3	
	Empyema without bronchopleural fistula		0	
	Incomplete or delayed re-expansion		2	
	Atelectasis		1	
	Recurrent spontaneous pneumothorax		0	
	Coronary occlusion		1	
	Wound infections		0	

The spread of the tuberculous infection is a danger in any operation for tuberculosis, yet we have noted only one case of contralateral spread and this was an increased activity of a previously noted disease probably because of a bronchopleural fistula, and the area of reactivation improved when re-expansion was obtained by re-establishing suction through an intercostal tube. The incomplete or delayed re-expansion of the remaining segments of the lung we believe is due to fibrosis from the healed processes in the remaining lung and is an indication for a relaxing type of thoracoplasty. The one death thought to be due to coronary insufficiency is probably a mortality of surgery in general rather than to the type of procedure performed.

The final evaluation of these cases of segmental resection for tuberculosis will require many years. All that can be said at this time is that the results thus far are such that we expect to continue using the procedure in carefully selected cases.

LUNG ABSCESS

Segmental resection for lung abscess (Table IV) is done only in the chronic forms of the disease or subsequent to drainage. However, resection was done in one case of acute putrid lung abscess. Pulmonary gangrene of the residual lobe occurred followed by a severe hemoptysis and death. At the present time, we believe that segmental resection should not be performed in cases of acute putrid lung abscess. The remaining twelve cases with chronic lung abscess were without complications. There were two cases in which resection was done with the tube still in place following drainage.

TABLE IV. SEGMENTAL RESECTION FOR LUNG ABSCESS

PATIENTS	OPERATIONS	SEGMENTS RESECTED	COMPLICATIONS	DEATHS
13	13	12	1	1
Complications in the Lung Abscess Cases				
	Pulmonary gangrene		1	
	Bronchopleural fistula		0	
	Empyema		0	

In a summary of the statistics there were 85 patients reported who had a total of 100 segmental resections requiring 91 operations with 30 complications reported, most of which could be considered minor. There were 3 deaths, or a mortality of 3.3 per cent, but only 1 of these deaths should be attributed to the type of operation performed.

DISCUSSION

Identification and division of the bronchus is mainly done to control secretions. By persistent effort and experience it is possible to become more and more adapt at this maneuver. Certainly the effort should be made in each case, and if ligation of a branch of the pulmonary artery facilitates early isolation of the bronchus, it should be done—especially in patients with tuberculosis. Identification of the bronchus is a matter of meticulous dissection. Prior to the operation, of course, roentgenograms, bronchograms, bronchoscopy, and laminag-

raphy allow the surgeon a preview of the task at hand. Since bronchograms are undesirable studies in patients with tuberculosis we have followed Klopstock's recommendation of using not only the posteroanterior, but lateral laminagraphic studies as well. The information gained by laminagraphy is actually more reliable than the surgeons' palpation. On occasion, at the operation we have changed some of our preoperative decisions and performed a lobectomy rather than a segmental resection. When this has occurred, postoperative examination of the surgical specimen more frequently coincides with the laminagraphic studies than with the surgeons' impression from palpation.

The *residual circulation* is significant in relation to the definition of segmental resection. Segmental resection, as generally used, is a term with an ambiguous meaning. In the past, and in this series, segmental resection has included the removal of the lingula, the superior segment of the lower lobe, and all the various segments of the other lobes. Nelson, Churchill, and Blades pointed out some time ago that the lingula and superior segment were essentially individual lobes and were occasionally separated from the other lobes by partial or nearly complete fissures. Blades more exactly reserved the term segmental resection for the removal of parts of a lobe. In principle, the technical removal of the lingula is quite different from removing one of its segments. For example, when the lingula is to be removed the cardinal structures including the vein are ligated at the hilum. On the contrary, if one of the segments of the lingula is to be removed the vein is not ligated at the hilum, because its main tributary is the intersegmental vein which receives blood from both segments. The latter procedure is more exactly called a segmental resection.

Obliteration of the pleural dead space by complete and immediate re-expansion is the most important single factor in the postoperative management. In those cases where good re-expansion was obtained the complications were few. The problems of re-expansion, assuming the bronchi are patent, depend upon, mainly: (1) the degree of pre-existing emphysema, (2) the duration of the disease, and (3) the amount of parenchyma to be removed.

Pre-existing emphysema, regardless of the etiological factor, is undesirable because such lungs are inelastic and already may be occupying a space closely approaching its maximum distensibility. Segmental resection rather than a lobectomy is particularly useful in these patients because the demand on the already enlarged lobes to fill a still larger space is, by comparison, a minor one.

The *duration* of the disease is significant in that the time factor influences greatly the compensatory factors at work. As the disease heals and contracts, the other segments often enlarge, especially if the disease occurred early in the patient's life. Removal, therefore, of the diseased segments imposes a minor demand upon the residual segments in obliterating the pleural dead space. The best results with the fewest complications may be anticipated in patients whose disease has been in existence since childhood.

The *amount* of lung removed greatly influences morbidity figures. If an upper lobe and superior segment of the lower lobe are removed, the demand upon the basilar segments to overdistend and fill the hemithorax is a major one. Add to this the hypothetical existence of extensive emphysema occurring late

in adult life when the compensatory factors are less effective and a most hazardous situation is created for the development of postoperative complications. In such cases it is often better to perform a thoracoplasty first, which tailors the thorax to fit residual segments.

The immediate morbidity rate of segmental resection is slightly greater than that of lobectomy. The operation takes longer to perform, demands a more meticulous dissection, and requires greater postoperative vigilance. But the late morbidity figures may prove to justify it because not only is the disease removed, but maximum pulmonary function is preserved.

CONCLUSIONS

1. The term segmental resection exactly used applies only to the removal of the bronchovascular components which combine to form a lobe.

2. The lingula and superior segment of the lower lobes are essentially individual lobes whose "primitive" fissure is usually undeveloped. The lingula is composed of two segments and the superior segment of the lower lobe is composed of three.

3. Preservation of the intersegmental vein in performing a segmental resection is a worthy refinement in technique.

4. Morbidity factors are reduced to the minimum by obliteration of the dead space through the complete and immediate re-expansion of the lung.

5. Continuous but controlled intrapleural suction lends the greatest assurance to the maintenance of normal physiologic re-expansion.

REFERENCES

1. Blades, Brian: Conservation of Lung Tissue by Partial Lobectomy, *Ann. Surg.* 118: 353-365, 1943.
2. Boyden, E. A., and Scannell, J. G.: An Analysis of Variations in the Bronchovascular Pattern of the Right Upper Lobe of Fifty Lungs, *Am. J. Anat.* 82: 27-74, 1948.
3. Churchill, E. D., and Belsey, R.: Segmental Pneumonectomy in Bronchiectasis, *Ann. Surg.* 109: 481, 1939.
4. Clagett, O. T., and Deterling, R. A., Jr.: Technique for Segmental Pulmonary Resection With Particular Reference to Lingulectomy, *J. THORACIC SURG.* 15: 227-238, 1946.
5. Nelson, H. P.: Postural Drainage of Lungs, *Brit. M. J.* 2: 251, 1934.
6. Overholt, R. H., and Langer, L.: New Technique for Pulmonary Segmental Resection; Its Application in Treatment of Bronchiectasis, *Surg., Gynec. & Obst.* 84: 257-268, 1947.
7. Swan, H., and Mulligan, R. M.: Experimental Study of Effect of Ligation of Pulmonary Veins in Dog, *J. THORACIC SURG.* 17: 44-56, 1948.

(See discussion on page 220.)

SEGMENTAL PULMONARY RESECTION

DETAILS OF TECHNIQUE AND RESULTS

RICHARD H. OVERHOLT, M.D.,* FRANCIS M. WOODS, M.D.,* AND
(BY INVITATION) BEATTY H. RAMSAY, M.D.†
BOSTON, MASS.

SINCE segmental resection of the lung was conceived by Churchill and Belsey,² this operative procedure has undergone neglect in childhood, enthusiasm and doubt in adolescence; it is now ready for acceptance of its maturity.

The development of segmental pulmonary resection is the result of the contributions of a succession of individuals. One of the first was the report of Nelson¹ who described each lung as having four basic parts, each of which was a separate anatomic unit. Later Alfred Goldman³ drew attention to the fact that a lung macerated in weakly corrosive solution tended to separate into segments. Churchill and Belsey applied the increasing anatomic knowledge to pulmonary resection and proposed and carried out segmental resections. Later contributions were made by Kent and Blades⁴ and Blades.⁵ Clagett⁶ reported a technique of lingulectomy wherein the intersegmental resection was commenced in the hilum and clamps were not used. He oversewed the pleural margins with interrupted sutures. Overholt and his associates⁷⁻¹¹ developed a technique of segmental resection and applied it to all segments and some subsegments. This technique was featured by blunt dissection of the intersegmental plane commencing in the hilum, avoidance of the use of clamps, and leaving the exposed intersegmental surface free to expand. Boyden¹² emphasized the intersegmental arrangement of the veins and many surgeons were aware of their presence in the intersegmental plane at the time of segmental resection. Betts¹³ pointed out this arrangement to Ramsay.¹⁴ The latter, following laboratory dissections, recommended that these veins be located in the hilum of the segment to be removed and followed as a guide to the intersegmental plane.

In its early development the procedure was largely but not entirely limited to lingulectomy by the clamp method. Abandonment of the clamp method and the persistent application of the individual ligation principle to all pulmonary segments by Overholt and his co-workers⁷⁻¹¹ featured the adolescent period. The discouragements at that time were principally the high incidence of complications; empyema occurred in 18 per cent⁷ and bronchopleural fistula in 6 per cent.⁷ These were the results of difficulties in the dissection of the intersegmental plane. They have been largely overcome by utilizing the intersegmental veins as guides to the plane following the suggestions of Ramsay.¹⁴ With continued experience both bronchopleural fistula and empyema are uncommon and unexpected.

Read at the Twenty-ninth Annual Meeting of The American Association for Thoracic Surgery, New Orleans, La., May 29-31, 1949.

*From the Department of Surgery, Tufts College Medical School. Member of Staff New England Deaconess and Pratt Diagnostic Hospitals.

†Formerly Fellow in Thoracic Surgery, Tufts College Medical School.

The purpose of this presentation is to record the details of operative technique as used today and the immediate postoperative results as found in the last twenty-eight consecutive segmental resections.

The steps of segmental pulmonary resection are essentially four in number. (1) The segmental artery is dissected out, ligated, and divided. (2) The segmental bronchus is cleared, ligated distally, divided, and the proximal stump closed. (3) The segmental vein, when present, is ligated and divided. (4) The segment to be removed is then separated from the lobe by locating the intersegmental vein in the hilum of the segment, and following this as a guide to the intersegmental plane. The first three steps are carried out in the order which is most convenient. The bronchus is located by palpation, the artery being adjacent. Care is taken to determine that they enter the segment to be removed. If subpleural veins, draining the diseased segment, are encountered, they are ligated and divided. Such veins are constantly found beneath the pleura of the lingula, the anterior segments of the upper lobes, and the middle lobe.

The arrangement of pulmonary veins can best be illustrated by considering the structure of the pulmonary unit or segment (Fig. 1). This we conceive is conical, covered by pleura, and contains an axially situated bronchus and artery. The principal veins lie on the surface and converge on the apex or hilum. There may be a small vein accompanying the artery and bronchus. When two or more of these units combine to form compound units or larger segments (Fig. 2) the surfaces in contact lose their pleural covering, and the subjacent veins become intersegmental in position. Elsewhere they remain as subpleural veins. Just as the bronchus and artery to a unit or segment are called segmental, so too is the vein which accompanies them. Thus, we have two types of veins: one central, accompanying the artery and bronchus and the segmental vein, and one peripheral; such a vein is either subpleural or intersegmental.

Finding the Intersegmental Vein.—The intersegmental vein is always to be found at the hilum of the segment in question and should be identified there rather than in the intersegmental plane. Distally there is danger that branching may have occurred and a branch followed between subsidiary segments. Just a thin covering of areolar tissue often keeps the intersegmental vein from view. Gentle dissection, either with scissors or a gauze pledget will readily expose it. Too much tension on the segment being removed will attenuate the vein, render it bloodless and white, and disguise it from recognition. The intersegmental vein usually bears the same anatomic relationship to the segmental artery and bronchus as does the principal lobar vein to the lobar artery and bronchus. Thus, in the upper lobe the intersegmental veins are generally anterior to the segmental arteries and bronchi, in the middle lobe anterior and inferior, and in the lower lobe posterior. Occasionally the intersegmental vein will receive the segmental vein as a tributary. A vein (Fig. 3), exposed in the hilum, may be a subpleural vein, a segmental vein, or an intersegmental vein leading between other segments than the ones to be separated. A subpleural vein can be seen to lie subpleurally on the surface of the lung. A segmental vein, when followed for a short distance distally, can be seen to accompany a bronchus. An intersegmental vein does not accompany an artery or bronchus. If it is the one

leading to the intersegmental plane to be dissected, it can be seen to run toward the junction between the diseased and the normal segments. This junction can be estimated from the configuration of the lung but is best demarcated by differential expansion of the diseased and healthy segments. Where the segment being removed adjoins more than one other segment, there usually will be a separate intersegmental vein for each separate surface (Fig. 4). Thus the posterior segment of the right upper lobe has an intersegmental vein and surface between it and the anterior segment and also between it and the apical segment. These two intersegmental surfaces form a V-shaped trough into which the posterior segment fits (Fig. 4).

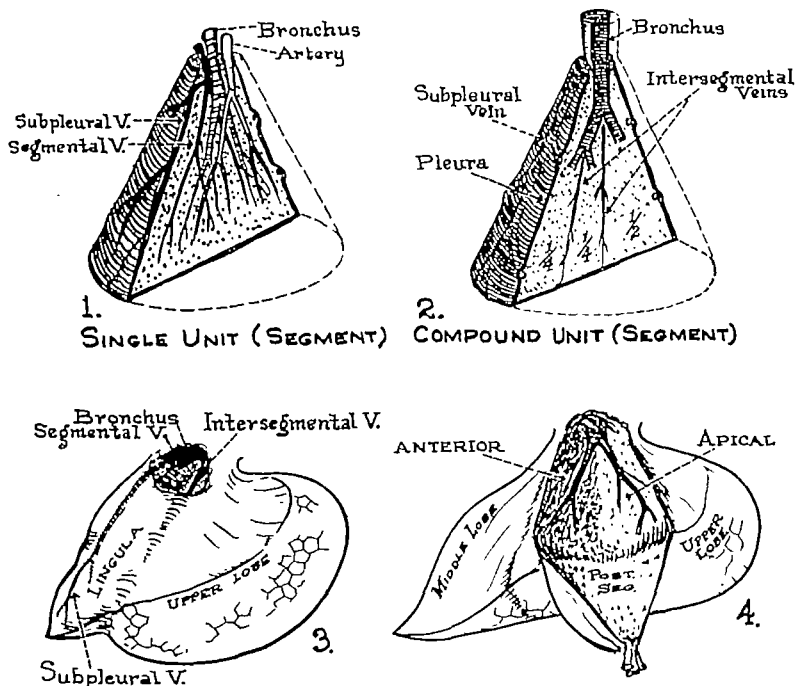


Fig. 1.—Our conception of the structural arrangement of the pulmonary unit. Note the central bronchus, artery, and segmental vein and the peripheral subpleural vein.

Fig. 2.—When such units are combined to form compound units or segments, some of the subpleural veins become intersegmental. The surgical segment illustrated here is shown divided into two quarter segments and a half segment corresponding to bronchial branching. Intersegmental veins demarcate the planes of separation.

Fig. 3.—A dissection of the hilum of the lingula: a subpleural vein is noted on the surface, a segmental vein accompanies a bronchus, and an intersegmental vein is shown between the lingula and the remainder of the upper lobe. Note that an intersegmental vein does not accompany an artery or bronchus.

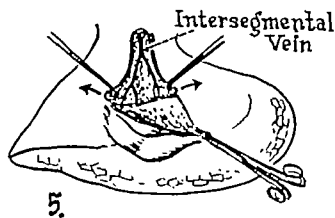
Fig. 4.—Segmental resection of the posterior segment of the right upper lobe is almost completed. Note the two intersegmental veins remaining on the freshly dissected surfaces of the anterior and apical segments.

After occlusion of the bronchus to the diseased portion, positive pressure is used to inflate the remaining lung, and then the contrast between the distended and collapsed parts outlines the approximate intersegmental plane. Frequently it is found that the diseased segment will fill with air although its bronchus has been occluded; in this situation deflation of the lung often will result in trapping of air within the diseased portion and provide the contrast in reverse.

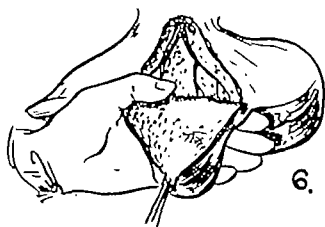
Separating the Surfaces.—In carrying out the separation of the diseased tissue, exposure and gentleness are paramount. Exposure is best obtained by irrigating the surface free from blood and by keeping it flattened out. If the edges are allowed to roll inward, an accurate dissection is almost impossible. The margins of the segment can be gently retracted by divergent pressure at two opposite points (Fig. 5). Inflation of the lung will flatten out the intervening surface. Once the plane of dissection has been partially developed, counterpressure from the pleural surface at the level of dissection is of distinct aid in supporting the tissues during the process of separation (Fig. 6). The dissecting motion should be a side-to-side sweep between the intersegmental vein and its branches on the healthy side and the diseased tissue on the other (Fig. 7). The surfaces separate easily unless there is considerable fibrosis. The pressure is directed parallel to the plane rather than at right angles to it and may be accomplished with the rounded end of small curved scissors or the finger tip. Small venous tributaries, arising in the diseased segment, and any fibrous strands are cut as encountered (Fig. 7). It has been determined by one of us (B. H. R.) that in the normal fresh autopsy lung it is a simple and speedy matter to insert two index fingers along the central axis of the intersegmental vein, to move these fingers divergently, and to separate cleanly the two adjacent segments (Fig. 8). This maneuver is successful also in clinical cases where the tissues of the intersegmental plane are normal. If any resistance is felt it is important to visualize and divide the cause which will be a fibrous band or a venous branch. Otherwise the finger may be deviated into the normal segment with resultant air leak. This was the reason for the single grade 2 fistula which developed in the present series. If there is considerable fibrosis in the intersegmental plane sharp dissection is necessary, all fibrous bands being precisely divided.

Sources of Error.—At the apex of the segmental cone the borders of the intersegmental plane are close together and the pleura here tends to resist the opening up of the plane. There is a temptation to start the dissection by incising the pleura and underlying parenchyma on each side. Division of the pleura alone may be of distinct help. Cutting the parenchyma is dangerous since bordering segments may be distorted by traction or by compensatory change, or may normally extend unexpectedly far down upon the side of the segment being removed. This is the case with the superior segment of the lower lobe which fits like a hood on the basal group with its posterior portion extending inferiorly over the basal segments.

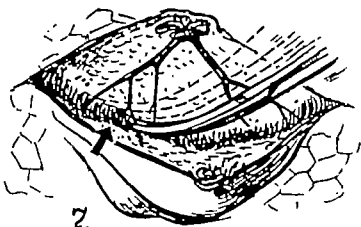
Damage to the lung surface is usually the result of "digging in" with the dissecting instrument or pulling on venous branches (Fig. 9). "Digging in" is the consequence of directing pressure against the normal segmental surface



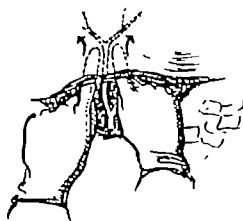
5.



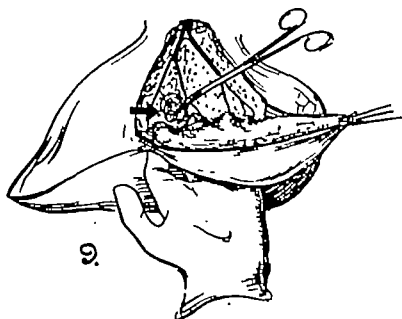
6.



7.

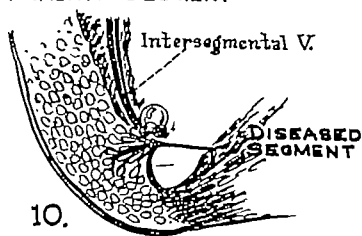


8.



9.

HEALTHY SEGMENT



10.

Fig. 5.—Dissection of the intersegmental plane is facilitated by flattening the plane out by divergent retraction of the edges and inflation of the lung.

Fig. 6.—As the plane is developed, counterpressure from the pleural surface aids dissection.

Fig. 7.—This illustrates the side-to-side dissecting motion and division of a venous branch.

Fig. 8.—If the tissues of the intersegmental plane are normal, segments can be separated cleanly by divergent motion of the fingers inserted along the intersegmental vein.

Fig. 9.—Alveolar damage and air leak are illustrated. An incorrect type of counterpressure is shown. Note the flat side of the hand in Fig. 6.

Fig. 10.—Undue traction on venous tributaries may be transmitted to the normal segment with resultant parenchymal tears.

and is more likely to occur if the finger tips are used for counterpressure. The areas of lessened resistance between the points of finger pressure allow the dissector to furrow the normal tissue. Counterpressure should always be produced by a flat surface (Fig. 6). Traction or pulling on the venous branches is transmitted to the tributaries entering from the normal segment; these are anchored in alveolar tissue which is torn and air leak results (Fig. 10). It is advisable to cut the small venous branches as they are exposed. Only the larger veins will require ligation.

The intersegmental vein between the superior segment and the basal group of segments of a lower lobe corresponds to ramus V^6b of Boyden.¹² It has two principal branches, one anterior and one posterior. Dissection along the posterior branch should not be done on its posterior aspect since this may cause damage to the overlapping portion of the superior segment. Its bronchus of supply, the posterior ramus of the superior segmental bronchus (ramus B^6c of Boyden), may be exposed (Fig. 11).

As the pleural surface is approached, there is again a temptation to cut through the remaining tissue. It is better to follow the veins to the pleura where they will be seen to arch over the surfaces of both segments (Fig. 12). Occasionally, if too much traction is used on the diseased segment, these veins may be stretched to a thin whiteness which makes them difficult to recognize. Relaxation of the traction will overcome this difficulty.

We know of no instance where a remaining portion of a lobe has undergone torsion following segmental resection. However, because of the theoretical possibility of such occurrence any adhesions or partial fusions of fissure lines between residual segments of one lobe and adjacent lung are left undisturbed (Fig. 13). Care is also taken at the time of closure of the chest that after the lung is inflated the relative position of the remaining lung is a normal one. As long as inflation is maintained throughout the convalescence no torsion should occur.

If the dissection is prolonged and continuous positive pressure is maintained, there may be a marked fall in blood pressure. In protracted dissections it is wise to interrupt the operation while the positive pressure is released.

Final Details.—While several dissections have been completed without a single air leak from the bare surface, there are usually one or two places which bubble slightly. If these are small and alveolar in origin they can be left safely to seal over in the first twenty-four hours. If the leak is more vigorous or if it is seen to be coming from an exposed bronchus, spontaneous closure cannot be depended upon. Attempts to suture these usually increase the amount of damage. Adequate control can be obtained by grasping the margins of the defect, with or without the adjacent parenchyma, between the teeth of fine forceps and by ligating the tissue so approximated (Fig. 14). All bleeding and significant air leaks having been controlled, nothing further is done to the surface.

To aid in re-expansion of the remaining lung all pleural attachments are separated. If the lower lobe or basal segments remain, the pulmonary ligament is divided. This permits the remaining lung immediately to rise higher within

the thorax, come in apposition with the pleural dome, and prevent apical accumulation of air or fluid. If full re-expansion of remaining segments is restrained by a pleural membrane, decortication should be practiced.

Prior to closure of the wound two catheters are placed intrapleurally. One is placed in the apex for the removal of air and brought out through the anterior portion of the incision. The other is placed just above the diaphragm for the

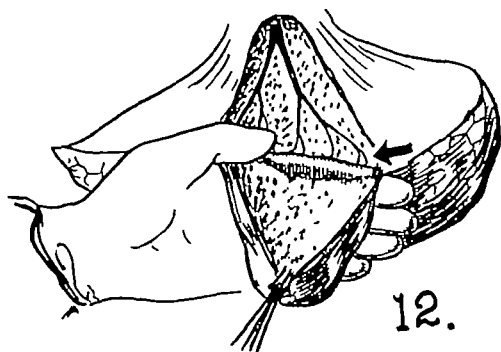
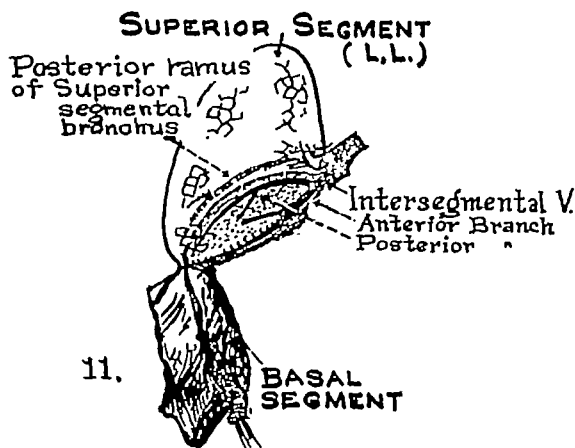
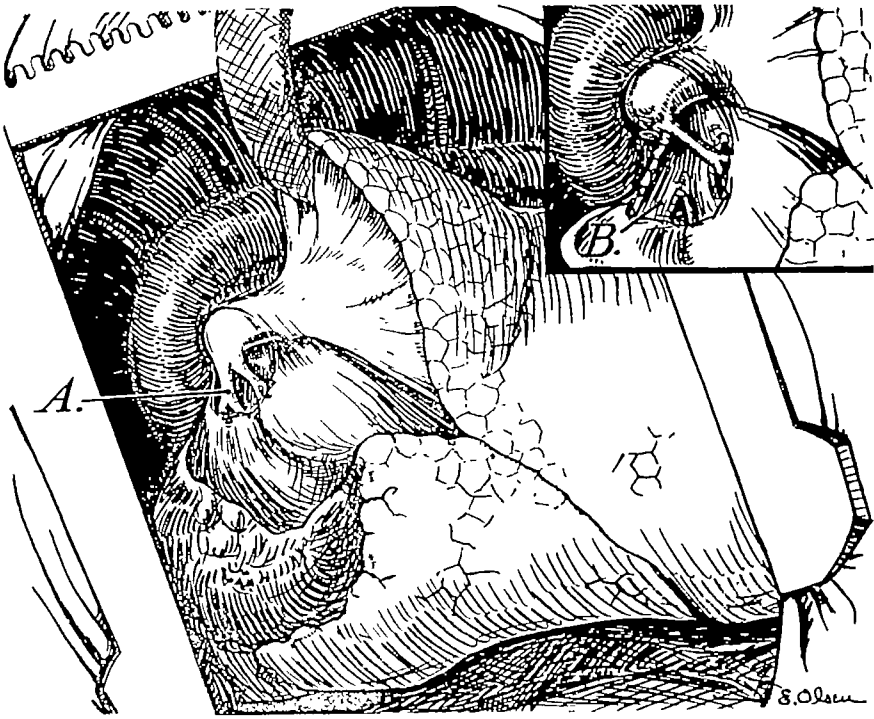
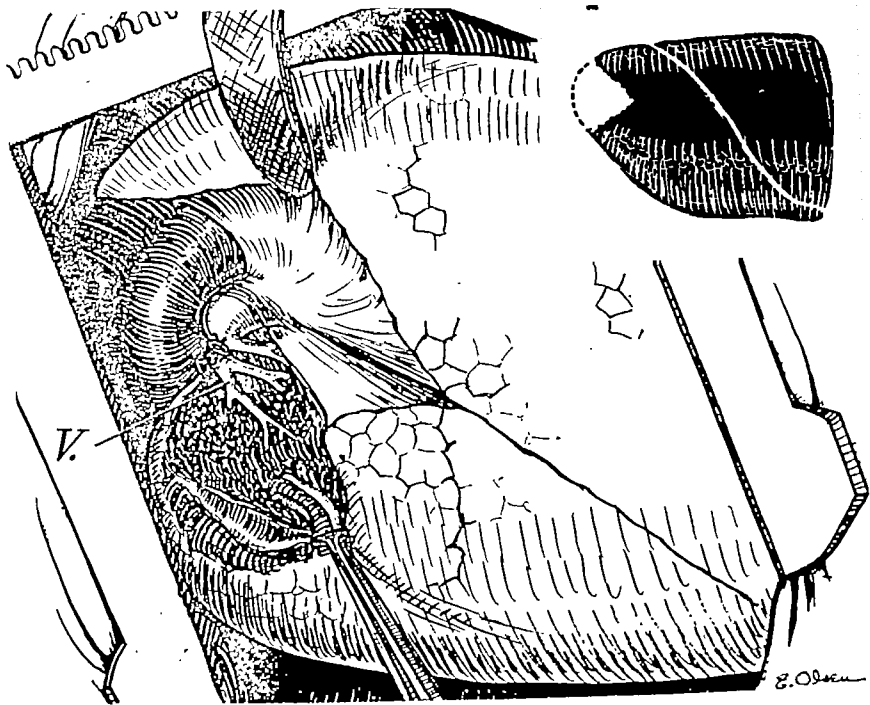


Fig. 11.—The posterior ramus of the superior segmental bronchus lies close to the intersegmental surface between the superior and basal segments of the lower lobe; it is easily injured.

Fig. 12.—The intersegmental veins are followed clear to the pleura before the latter is divided.



A.



B.

Fig. 16, A.—Resection of the apical segment of a left upper lobe. The artery (A) to the apical segment is exposed. The branch to the posterior segment is also seen.

Insert: The artery is divided. The bronchus (B) to the apical segment is exposed and the posterior segmental bronchus is also visible.

Fig. 16, B.—The bronchus is divided and dissection of the intersegmental plane is almost complete. Note the intersegmental veins (V).

Insert: Diagram showing left lung after resection of apical segment.

In fifteen cases the lung was completely re-expanded and both catheters were removed within the first twenty-four hours, in ten within forty-eight hours, and in the remaining two on the third and fifth days, respectively. This last patient had an upper lobectomy with segmental resection of the superior division of the lower lobe for tuberculosis. The residual lower lobe was not decorticated to prevent overexpansion; thoracoplasty was to follow. One patient died post-operatively.

The average postoperative hospitalization was 14.0 days. The shortest period was ten days and the longest nineteen days. The discharge date varied somewhat depending upon the distance to be traveled. Formerly the hospital stay averaged three weeks.

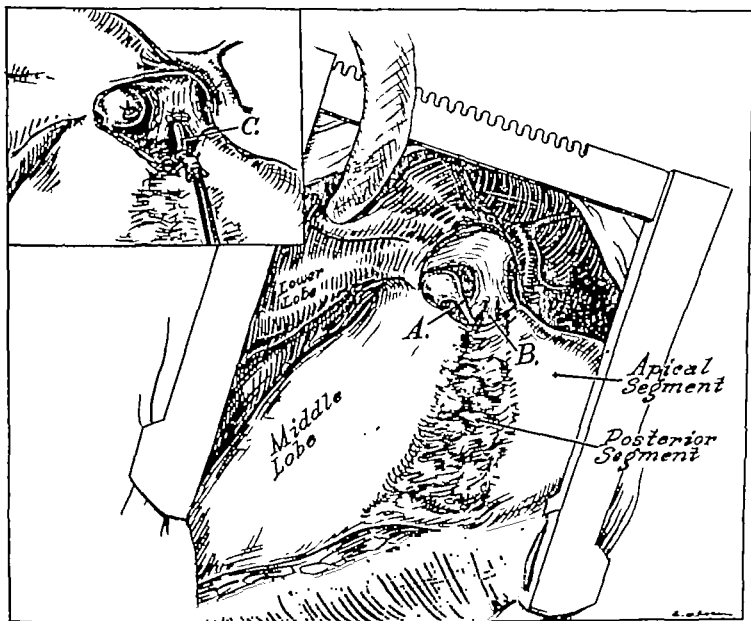


Fig. 17, A.—Resection of the posterior segment of a right upper lobe. An accessory artery to the posterior segment (A) is exposed and the bronchus (B) to the upper lobe and its three branches are exposed.

Insert: The bronchus and accessory artery to the posterior segment are divided. The posterior segmental artery (C) is now exposed lying anterior to the divided bronchus.

Table III lists the complications. There were no empyemas, two minor fistulas, and one death. Fistulas are graded first degree if the leak is so small that it is easily controlled by aspiration, second degree if it requires the reinsertion of an intrapleural catheter to obtain re-expansion, and third degree if it requires operative closure with or without decortication of the remaining lung tissue. The one second-degree fistula closed and the lung re-expanded

within twenty-four hours following reinsertion of an intrapleural catheter which was connected for suction. The one death occurred from pulmonary insufficiency aggravated by inability to raise the bronchial secretions.

TABLE III

COMPLICATIONS		
Empyema		0
Fistula		2
1st degree	1	
2nd degree	1	
3rd degree	0	
Death		1
Engorgement of superior segment		2
Total		5

This 24-year-old girl had had the middle lobe, basal group, and anterior segment of the upper lobe removed from the right lung on March 23, 1948. On October 11 the basal group and lingula were removed from the left lung. Postoperatively she was cyanotic in spite of re-expansion of the remaining lung on the left and no evidence of air leak from the intersegmental surfaces. Four

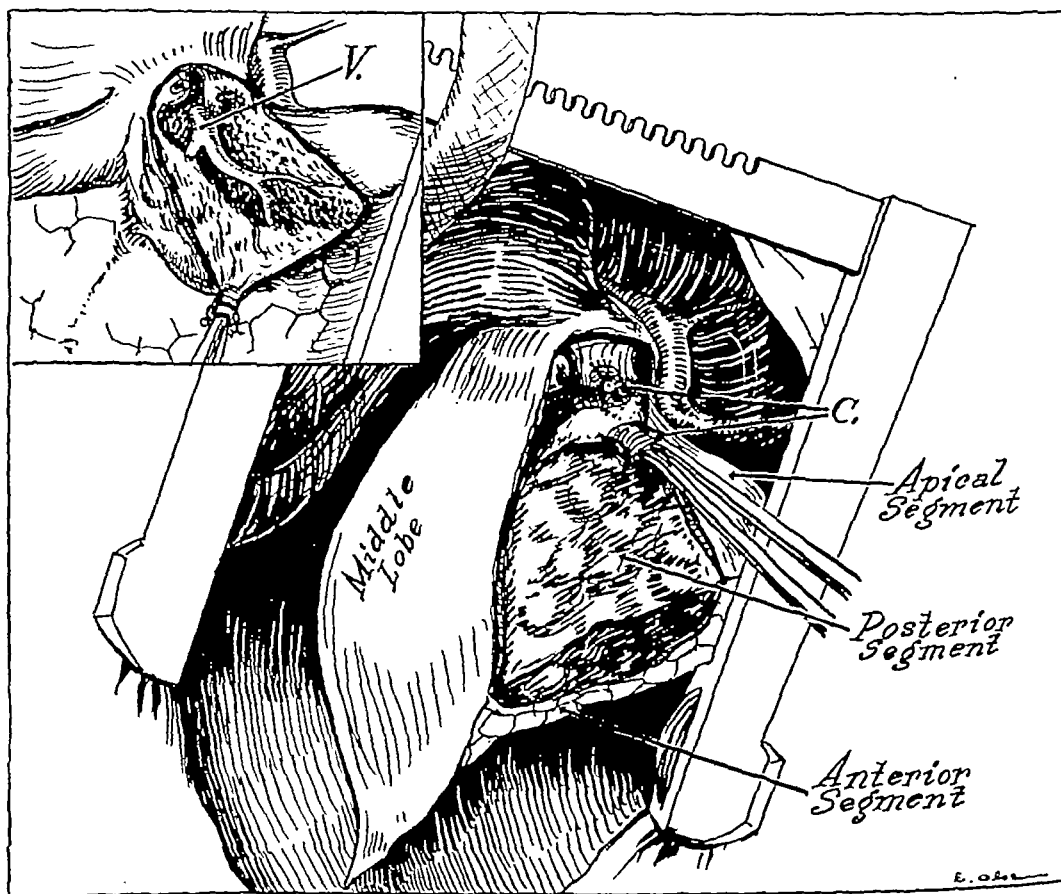


Fig. 17, B.—The posterior segmental artery has been divided. The intersegmental vein is obscured by areolar tissue. Note that the middle lobe and upper lobe are fused in this instance. Insert: Dissection of the intersegmental plane between the posterior segment and the apical segment is completed. Dissection of the plane between the posterior segment and the anterior segment is just started. Note the intersegmental veins (V) remaining on the intersegmental surfaces.

hours postoperatively she had sudden difficulty in breathing. Although tracheal aspiration was successful in relieving her, she almost immediately had a second attack and succumbed before aspiration could be carried out. Autopsy revealed bronchiectasis in all but one of the remaining pulmonary segments and less than $\frac{1}{2}$ c.c. of thin secretion in the left bronchial tree. The superior segment of the lower lobe was engorged with blood. The intersegmental surfaces were dry and no air leak could be demonstrated.

The engorgement of the remaining superior segment of the lower lobe following segmental resection of the basal group is a complication never recognized by us previously. This was not accompanied by necrosis. One other patient had a slight postoperative fever associated with an increase in density of the remaining superior segment as demonstrated by roentgenogram. These signs spontaneously disappeared in twelve days. We believe that this may represent a second case of engorgement. It is considered likely that this complication is produced by obstruction of the venous drainage from the superior segment. Retraction of this vein or torsion of the lobe, produced by attempts to obtain exposure of the basal bronchi, may obstruct it for a varying period

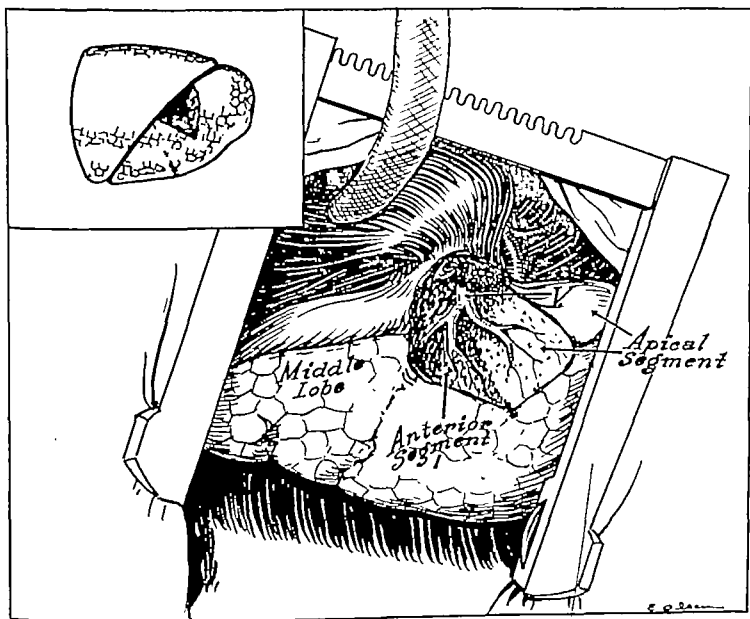


Fig. 17, C.—Posterior segment is completely removed, intersegmental veins (V) remaining on both the intersegmental surfaces.

Insert: Diagram showing right lung after resection of posterior segment.

of time (Fig. 15). Since the complication has been recognized we have carried out the mobilization of the basal bronchi from the anterior and inferior aspects as much as possible and have taken care not to obstruct the vein in question.

The value of segmental resection was emphasized by the anomalous condition found in a 10-year-old girl. She had bronchiectasis limited to the lingula and basal segments of the left lower lobe. At operation she was found to have a lung consisting of a single lobe without any trace of a fissure. It was not possible to expose the segmental arteries in the fissure as is customary, but by dividing the segmental bronchi and veins first, the arteries were dissected free without difficulty; then the intersegmental veins were located and followed as guides to the two intersegmental planes. No air leak came from the bare surfaces; the remaining lung re-expanded almost immediately and the intrapleural catheters were removed in less than twenty-four hours. She was discharged on the thirteenth postoperative day.

SUMMARY

The details of pulmonary segmental resection, with special attention to dissection of the intersegmental plane, have been described and illustrated. Emphasis is placed on using the intersegmental vein as a guide to the plane. There has been a marked reduction in complications.

REFERENCES

1. Nelson, H. P.: Postural Drainage of Lungs, *Brit. M. J.* 2: 251, 1934.
2. Churchill, E. D., and Belsey, R.: Segmental Pneumonectomy in Bronchiectasis, *Ann. Surg.* 109: 481, 1939.
3. Goldman, Alfred, quoted by Churchill, E. D.: Symposium on Resection of Lung (E. Starr Judd Lecture), *Surgery* 8: 961, 1940.
4. Kent, E. M., and Blades, B.: The Anatomic Approach to Pulmonary Resection, *Ann. Surg.* 116: 782, 1942.
5. Blades, B.: Conservation of Lung Tissue by Partial Lobectomy, *Ann. Surg.* 118: 353, 1943.
6. Clagett, O. T.: A Technique of Segmental Pulmonary Resection With Particular Reference to Lingulectomy, *J. THORACIC SURGERY* 15: 227, 1946.
7. Overholt, R. H., and Woods, F. M.: Segmental Pulmonary Resection for Bronchiectasis, *South. Surgeon* 13: 777, 1947.
8. Overholt, R. H., Woods, F. M., and Betts, R. H.: An Improved Method of Resection of Pulmonary Segments, *J. THORACIC SURGERY* 17: 464, 1948.
9. Overholt, R. H., Betts, R. H., and Woods, F. M.: Multiple Segmental Resection in the Treatment of Bronchiectasis, *Dis. Chest* 13: 583, 1947.
10. Overholt, R. H., and Langer, Lazaro: A New Technique for Pulmonary Segmental Resection, *Surg., Gynec. & Obst.* 84: 257, 1947.
11. Overholt, R. H., and Langer, Lazaro: *The Technique of Pulmonary Resection*, Springfield, Ill., 1949, Charles C Thomas, Publisher.
12. Boyden, E. A.: The Intrahilar and Related Segmental Anatomy of the Lung, *Surgery* 18: 706, 1945.
13. Betts, R. H.: Personal communication.
14. Ramsay, B. H.: The Anatomical Guide to the Intersegmental Plane, *Surgery* 25: 533, 1949.

DISCUSSION ON "SEGMENTAL RESECTION IN PULMONARY DISEASES" BY
DR. J. MAXWELL CHAMBERLAIN AND COMMANDER THOMAS C. RYAN,*

AND "SEGMENTAL PULMONARY RESECTION" BY DR. RICHARD H.
OVERHOLT, DR. FRANCIS M. WOODS, AND DR. BEATTY H. RAMSAY

DR. PAUL C. SAMSON, Oakland, Calif.—This is, of course, a very interesting experience to which we have listened. Dr. Dugan and I would like to present briefly our

*See page 199 for article by Chamberlain and Ryan.

results during the last three years with approximately seventy-five anatomic segmental resections, including bisegmental removals. Several points I believe are of importance; we have not attempted to split the middle lobe segmentally, believing that removal of the middle lobe in toto probably does not sacrifice too much lung tissue even though one segment is involved. We have had occasionally some difficulty in attempting to determine both preoperatively and at operation whether one or several basal segments were involved, and so in general we have performed a combined basal segmental resection. On several occasions we have removed subsegments which seemed to be worth while, and I would like to show two slides to illustrate this. (Slides.)

These lateral films were taken one year apart. In the interim resection on the left side was performed. The original bronchogram showed middle lobe disease and a small amount of disease in the anterior ramus of the anterior segment of the upper lobe. One year later there appears to be some improvement in the middle lobe although there is as much bronchiectasis in the anterior ramus, the small puddle of Lipiodol being in the anterior subsegment. Here is the small lymph node often seen, causing some compression and stenosis, and here the atelectatic portion of the anterior subsegment. The dissection continued with the anterior subsegmental bronchus divided and the lateral subsegmental bronchus left intact.

We have closed the pleural surface because we have had too many complications, we believe, by leaving a raw lung surface. This is performed with little or no actual diminution in breathing capacity, because we do it with the lobe partly expanded, leaving a small rim of pleura which can be brought over.

Here is a patient who has bronchiectasis; this shows the first bronchogram with superior segmental disease, but two areas which are not filled. Further bronchograms were necessary, which showed disease in the posterior upper lobe segment and also in the middle lobe. At operation the distribution was right middle lobe, the superior segment of the right lower, and lateral subsegment of the posterior segment of the right upper. These three areas of disease were removed, resulting in considerably better functional result than if entire lobes were removed.

In conformity with other experience, our complications have been definitely greater, approximately three times greater in the segmental resections than in lobar resections. In particular, empyemas have caused us some difficulty. Whereas in lobectomy there were approximately 3 per cent complications, in the first thirty segmental resections in which a raw lung surface was left we had a 23 per cent rate of empyema, an incidence which we believe to be too high. For that reason we have tended to close practically all raw lung surfaces and in the last 40 odd cases, empyema has dropped to about 5 per cent.

DR. ROBERT R. SHAW, Dallas.—I have enjoyed these two papers very much. Dr. Paulson and I want to report our experience in this field in the last three years. During this period we have done segmental resections for nonmalignant disease in the lung in eighty-four instances and have had a general empyema rate in this whole group of a little more than 18 per cent. This included, however, twenty-two segmental resections done for abscesses and infected cysts. In this group the empyema rate was quite high—34 per cent. If we limit the discussion to patients having bronchiectasis and chronic pneumonitis, there were 60 segmental resections with an empyema rate of approximately 13 per cent. This contrasted with pneumonectomies or lobectomies for the same condition with an empyema rate of 2.7 per cent in 106 instances. There is one other technical aspect of the problem that I would like to emphasize; that is the fate of the remaining segment. The fate of the remaining segment disturbed me considerably early in our experience, especially after the basal segments had been removed from a lower lobe. I noticed on postoperative bronchoscopy that there was kinking of the dorsal bronchus by dislocation of the dorsal segment. Since that time I have routinely left adhesions if they were there, to allow this segment to come down uniformly with the upper lobe in the re-expansion process. If there were no adhesions I made a row of mattress sutures, picking up just the visceral pleural edges so that the dorsal lobe would expand evenly with the upper lobe.

I would like to show one slide to illustrate a problem brought up by segmental resection. In the patient with bilateral bronchiectasis, many more of course are being accepted for surgery now than could have been accepted before segmental resection had become safer. I think the choice of the side to be attacked first sometimes becomes a great problem. We have here an illustration of a woman who has bronchiectasis of the lower segments of the right lower lobe; the lateral film shows that the dorsal segment is normal; there is bronchiectasis complete in the middle lobe, and bronchiectasis in the anterior segment of the upper lobe. To attack the right side first entails a rather difficult dissection and also invites complications that may lead to poor function of the remaining segments in this lung. If a poor function did result on this side it might be impossible to do anything about the lingular segment, which is the only portion involved on the left side. I think it would be better wisdom in a case of this sort to attack the lesser side first, since we would have a functioning right lung—although it is damaged by disease—to give support during the attack on the small lingular segment. I think the left side could be handled easily without too much danger from the operation. Then, with a left lung free of disease, I think the right side could be attacked much more safely. In a later paper you will note that the four deaths occurring in bilateral bronchiectasis occurred after the attack on the second side, and quite often these deaths are due to insufficient pulmonary reserve. If technical difficulties should be encountered on the right side, as sometimes occurs—sometimes an artery is torn so that more tissue has to be removed than was planned—even a pneumonectomy could be performed on the right side and the result would probably be good. If such a thing should occur by attacking the right side first, nothing could ever be done about the disease on the left. It might take a bit of salesmanship to sell the patient the idea of attacking the lesser side first, but I think there are instances in which this would be good judgment.

DR. D. A. MULVIHILL, New York.—I think Dr. Chamberlain has been altogether too modest in glossing over in half a sentence the thirty cases of segmental resection he has done for pulmonary tuberculosis. He has undertaken this study as part of an experimental project at the Triboro Hospital in New York and the U. S. Naval Hospital at St. Albans, N. Y. I have had an opportunity to observe his results to date on this work. He is naturally reluctant to talk about them because he feels, I think, that his indications for segmental resection in tuberculosis are not adequately defined at present and he would like to work them out better. It is true, as he mentioned, that it will take five years to evaluate the final results in these cases. However, thirty cases of segmental resection for pulmonary tuberculosis at this date is no small number. I can say that his results, so far as the early period is concerned, are most promising. He has a low rate of immediate postoperative complications, and so far very few and insignificant late complications in the months that have passed since operation on this group of segmental resections. I think probably he should speak more freely about his experience with this particular group when he closes the discussion.

DR. R. P. GLOVER, Philadelphia.—I should like to re-emphasize one point in the technique of segmental resection as this procedure is applied in tuberculosis. Whereas in nonspecific inflammatory segmental resections we have had excellent success using the technique described by Dr. Clagett, that of leaving the raw surface open and unencumbered by pleura, in tuberculosis the multiple so-called alveolar leaks which frequently occur after segmental resection must be treated with more respect. These leaks ordinarily quickly seal themselves, but in a series of some thirty odd segmental resections in pulmonary tuberculosis we have found that such leaks not infrequently do not become sealed and therefore lead to nonexpansion of the lung. I should like to suggest, therefore, that very meticulous care be taken in tuberculous segmental resections to find and close these leaks, which actually may be tiny bronchiolar openings rather than so-called alveolar tears. These openings must be carefully closed, either by the technique described here or by the

use of a flap of pleura. If this precaution is not taken, proper expansion of the lung will not occur, almost invariably leading to the formation of infected pleural fluid and eventual empyema with bronchopleural fistula. Attention to this detail will give better results in tuberculous cases treated by segmental resection.

DR. J. D. MURPHY, Oteen, N. C.—A few weeks ago at a meeting in North Carolina I was seated next to Dr. Stuart Willis, and the subject of segmental resection in tuberculosis arose. Dr. Willis said it was a vicious procedure and that the results with it were bad. He asked whether we were doing it at Oteen. I was rather noncommittal but when I got home I went through our cases and found that in a series of 110 resections for tuberculosis we had done segmental resection in 9 instances. These varied from a formal dissection of the dorsal segment or an apical segment to simple ligation and excision of the tip of the dorsal segment as described by Dr. Bloch in 1881.

In analyzing the results in these patients, eight had a negative sputum, and re-expansion without complications. One had late progression of the disease into the remaining segment of the upper lobe and required thoracoplasty. All have been done for at least six months and some of them for eighteen months.

DR. FELIX HUGHES, JR., Memphis.—This slide shows the distribution of resections of the lung at Kennedy Hospital since June, 1946. In tuberculosis we have done seventeen segmentals, in bronchiectasis seventeen, including the patients who had both a lobe and a segment removed; for abscess and pneumonitis we have done two segmental resections; for cysts or blebs we have also done two segmentals. In addition, I have listed, under "lesion," patients who have not had a formal segmental resection but have had an excision; there are fifteen of those. There has been no mortality in the group with benign lesions.

In the patients with tuberculosis our results have been more an indication of the stability of the lesion than of the type of resection. Where the patient had a stable lesion, 80 per cent are in good condition at this time, most of them seven to fifteen months after operation. Where the lesion was acute or unstable, only 25 per cent are in excellent condition at this time, and that has been about the same regardless of whether the operation was a lobectomy or a segmental resection. Of the forty-three patients with pulmonary tuberculosis who have had a resection only one patient is in serious condition at this time.

The slides will show the result in two cases. This is a Negro patient with a tuberculous lesion in the right upper lobe, treated by streptomycin. While streptomycin was being given the process spread to two basal segments and the apical segment of the right lower lobe. Lobectomy and segmental resection were performed, with streptomycin protection, and, following this, recovery has been uneventful. Thoracoplasty was done at the end of three weeks, and this slide shows the result after thoracoplasty and resection. It has been over one year since the last operation, and the sputum is still negative.

The next slide shows a patient who had bilateral pulmonary tuberculosis; he had a pneumothorax on the left side and pneumonolysis with complete division of adhesions, but the disease progressed with cavity rupture, a bronchopleural fistula, and total empyema on the left. In addition, therapeutic pneumothorax was instituted on the right, with the diseased area becoming considerably fibrosed during streptomycin therapy. Decortication was done with segmental resection of the posterior apical segment of the left upper lobe, followed, after three weeks, by thoracoplasty. The next slide shows the condition soon after thoracoplasty with operative edema still present. Since that time, this has cleared and the anterior segment is expanded. The sputum is negative and the left lung remains clear. There is a suspicious lesion on the right, but the patient's condition is good.

I agree with Dr. Chamberlain that it is too early to decide the value of segmental resection in tuberculosis, but it is certainly worth considering if we can remove all the diseased tissue and still conserve good lung tissue.

DR. JOHN M. CHAMBERLAIN, New York.—I do not believe that I can over-emphasize the vigilance which these patients require postoperatively. When Dr. Samson and Dr. Shaw reported their empyema rate I was rather disturbed and hurriedly went back over my statistics to see if I had been inaccurate in my evaluation. I found that we have recorded one case of empyema in the series, but under a separate heading we have recorded six cases of bronchopleural fistula and under another heading three cases of recurrent or spontaneous pneumothorax. If all are considered to be empyemas, the empyema figure adds up to ten. Yet only one of them developed the clinical picture of an empyema and all of them responded to intercostal decompression (tube) done in the patient's bed without returning to the operating room. I should like to point out that perhaps the great difference in our results is due to postoperative intrapleural active suction. At Bellevue, on Dr. Berry's service, there are eight of us, and when we first began to perform this operation there were two schools of thought as to whether a water seal was sufficient or whether we should use active suction. I believe I can say that most of the men now believe that active intrapleural suction is a better solution.

We have used in one case as high as six intercostal tubes postoperatively (not all at once of course) in instances where localized pockets were found in the postoperative x-ray films. We feel that the dead space is a potential empyema and that its obliteration prevents the development of an empyema. Therefore any intrapleural pockets seen in the early postoperative course of these patients are "tubed" immediately and the tube is left in for twenty-four to forty-eight hours. We wish to point out that if the empyema rate is high it may be because the surgeon failed to insert a tube and re-expand these small pockets which may occur. Perhaps the difference in our results is dependent upon one's definition of what is and is not an empyema.

If I were going to close the surface of one of these segmental resections I cannot imagine a more ingenious way than the one which Dr. Samson has shown us. I have thought for a long time of some way of using a pleural graft, as I saw Dr. Churchill do years ago over the raw area of the mediastinum after a pneumonectomy. Dr. Samson's solution is a good one because as the visceral pleura is approached it should be very easy to skim the visceral pleura off the diseased segment and allow it to fall back over the raw area as he pointed out. However, the collection of air beneath this pleural graft might prevent it from closing the small fistulas. I prefer to rely on suction which removes the air faster than these leaks can deliver it.

I appreciate all that the various discussors have contributed. With reference to tuberculosis I am still not anxious to draw any conclusions. It might interest you to notice the complications we have listed in those 30 cases. Contralateral spreads have occurred in one case; homolateral spreads have occurred in two cases; bronchopleural fistula in three cases; empyema without bronchopleural fistula in none (two have developed since this paper was presented); incomplete or delayed re-expansion in two cases; atelectasis in one case; recurrent spontaneous pneumothorax in none; coronary occlusion in one case; wound infection in no cases.

I do not think anyone can feel that segmental resection in tuberculosis is a matter that can be decided on today, and with Dr. Murphy and the doctor to whom he referred I agree that it is a very hazardous procedure and, first of all, the surgeon should master the technique of segmental resection and then, with the aid of the medical service, consider very cautiously and select very carefully those cases which might be vulnerable to this type of procedure.

DR. FRANCIS M. WOODS, Brookline, Mass.—We agree heartily with what Dr. Chamberlain has said about active suction; we have found that active suction rather than just underwater-seal drainage is definitely more advantageous in getting prompt and complete re-expansion of the lung.

Regarding Dr. Shaw's remarks about the selection of the side for first operation in bilateral bronchiectasis, I would not agree with him because it seems to me that the most important consideration is the postoperative care of the patient and when the side of the

greater bronchiectasis is left in, we often have great difficulty in managing the aspiration and coughing up of secretions. Therefore I would feel at the present time that operating on the side of the greater bronchiectasis is usually more advantageous.

(Slides). This illustrates a complication that Dr. Ramsay did not go into; we have found in two instances, we believe (certainly one was proved at autopsy), that there was considerable engorgement of the superior segment of the lower lobe after the basal segments had been removed. We attribute this, as we attempt to demonstrate in this diagram, to the fact that during exposure of the bronchus to the basal segment of the lower lobe, undue traction or too prolonged traction was placed on the vein to the superior segment. We therefore recommend and practice at the present time, approaching the bronchus from the inferior or anterior side rather than the posterior side, so that undue and prolonged traction will not be placed on that vein.

I would also like to mention that we have become interested in another application of segmental resection. When the chest is opened and the exact diagnosis has not been established, sometimes one finds it difficult to get an adequate biopsy without cutting through lung tissue. In these instances we have done segmental resection to establish the diagnosis. This case turned out to be a carcinoma, and pneumonectomy was completed. The next slide shows a tuberculoma removed by segmental resection. Frozen section established the diagnosis and the chest was closed.

BRONCHOLITHIASIS

HERBERT W. SCHMIDT, M.D.* (BY INVITATION), O. THERON CLAGETT, M.D.,†
AND JOHN R. McDONALD, M.D.‡
ROCHESTER, MINN.

THE subject of broncholithiasis has been of great interest to the medical profession for centuries. It has been said that pulmonary calcifications interested Aristotle (384-322 B.C.).¹ However, a detailed description of the condition did not appear until 1600 A.D. when Schenck a Grafenberg² described 17 cases of broncholithiasis reported in the literature. Reports of an additional 6 cases were reviewed by Schenck a Grafenberg, but from the text it is impossible to be certain that the authors were dealing with bronchololiths.

Morton,³ in 1689, outlined therapy for broncholithiasis. He stated:

First; in the beginning of the disease lubricating medicines should be used copiously; yes, and mild emetics, by which one attempts to promote the expulsion of the calculus or of other bodies that have lodged in the lungs, since long delay makes it more difficult to expel them and the condition of the lungs is thence more gravely injured. Secondly; if, however, severe and spasmodic pains are excited by the laceration of the lungs caused by motion of prickly calculi of this kind, copious hemoptysis is to be expected; and therefore instead of lubricating medicines laudanum should be given freely and frequently, by which the motion of the calculus can be stopped promptly, pain can be relieved and hemoptysis can be checked. . . . Certainly if phthisical patients of this kind take care of their health, as by always breathing pure country air, avoid taking cold, drinking bouts, excessive exercise and whatever else can excite the fixed calculi to new movement, they will be able to live to an advanced old age, performing their usual duties satisfactorily. . . . Only when there is urgent need should a milk diet be ordered in phthisis arising from pulmonary calculi, because such a diet is likely to generate new chalky stones of this kind in the lungs. Whence it happens that the cure of one phthisis is accustomed to lay the foundation for another.

He reported 2 cases of broncholithiasis.

Boerhaave,⁴ in 1744, described the case of the botanist, Sebastian Vaillant, who, through many years, coughed up 400 bronchololiths and died at 54 years of age of "phthisis calculeuse."

Morgagni¹ stated that "almost 100 authors have described calculi, or topi, that have been brought up by coughing, or found in the lung." He did not indicate how many of these were bronchial calculi. He again brought up the subject as to whether milk should be used in patients who had broncholithiasis. He agreed with Morton, feeling that it was contraindicated. He quoted other authors, among them an Albertus Fabrius, who contended "very resolutely" that calcareous concretions in the lung might be increased by the use of milk. He cited the case of a patient who craved milk "to such an extent that he drank it even instead of water or beer." It was felt that this program

Read at the Twenty-ninth annual meeting of The American Association for Thoracic Surgery, New Orleans, La., March 29-31, 1949.

*Division of Medicine, Mayo Clinic.

†Division of Surgery, Mayo Clinic.

‡Division of Surgical Pathology, Mayo Clinic.

"increased the calculus, or at least the pain, and hastened the patient's death." Another patient described by Fabrius was refused milk in the diet and given oil of almonds. A broncholith was coughed up, the pain left, and "perfect health was restored."

In 1810, Bayle⁵ described stonelike phthisis as being one of the six types of tuberculosis.

By 1892, microscopic and bacteriologic techniques had been perfected to such an extent that Legry⁶ was able to approach the problem from this standpoint. He stated that calcification of tracheobronchial lymph nodes could occur in tuberculous as well as nontuberculous patients. He pointed to the fact that certain workers as early as 1854 had held to this opinion but that their work had not been readily accepted. He stated that tubercle bacilli could be found in only a certain small percentage of the cases in which he studied specimens microscopically. Studies on guinea pigs gave negative results. It was his opinion that this was one way that nature could get rid of a tuberculous lesion, namely, by first having a concretion form and then by having it coughed up. He advised the giving of milk and of calcium phosphate to tuberculous patients since here were substances which would cause a "fossilisation of bacilli and their loss of virulence."

REVIEW OF LITERATURE

Incidence.—The exact incidence of broncholithiasis is difficult to determine because (1) not all of the recognized cases have been reported and (2) not all cases have been recognized. Stivelman⁷ stated that the incidence of broncholithiasis in pulmonary tuberculosis is exceptionally low; he saw only 1 instance in more than 5,000 cases of tuberculosis. Pritchard⁸ recorded 2 examples as having occurred in 7,000 patients with tuberculosis. Lloyd⁹ was able to find reports of only 18 cases of broncholithiasis in the English medical literature from 1900 to 1930. He reported 4 cases of his own. By the end of 1941, VanOrdstrand, Moore, and Harris¹⁰ were able to find reports of 26 cases in the English medical literature. They reported 2 cases at that time. In August, 1944, Tinney and Moersch¹¹ stated that 30 cases had been reported in the British and American literature. They pointed out that this did not represent the real incidence of broncholithiasis since they were able to collect records of 28 cases from the files of the Mayo Clinic.

Sternberg,¹² in a study of 6,132 post-mortem examinations, observed 32 instances in which there was evidence of perforation into the tracheobronchial tree by an anthracotic lymph node.

By the latter part of 1946, Zahn¹³ was able to find reports of 71 cases in the literature. He added another case. His was the only case of broncholithiasis encountered in more than 4,000 cases of tuberculosis during the year 1945 at Fitzsimons General Hospital. Lell,¹⁴ in 1947, reported another 6 cases.

In the previously mentioned reported cases, the diagnosis was made by (1) obtaining a history of having coughed up a broncholith, (2) finding a broncholith at the time of bronchoscopy, (3) surgical exploration, or (4) post-mortem examination.

✓

BRONCHOLITHIASIS

HERBERT W. SCHMIDT, M.D.* (BY INVITATION), O. THERON CLAGETT, M.D.,†
AND JOHN R. McDONALD, M.D.‡
ROCHESTER, MINN.

THE subject of broncholithiasis has been of great interest to the medical profession for centuries. It has been said that pulmonary calcifications interested Aristotle (384-322 B.C.).¹ However, a detailed description of the condition did not appear until 1600 A.D. when Schenck a Grafenberg² described 17 cases of broncholithiasis reported in the literature. Reports of an additional 6 cases were reviewed by Schenck a Grafenberg, but from the text it is impossible to be certain that the authors were dealing with broncholiths.

Morton,³ in 1689, outlined therapy for broncholithiasis. He stated:

First; in the beginning of the disease lubricating medicines should be used copiously; yes, and mild emetics, by which one attempts to promote the expulsion of the calculus or of other bodies that have lodged in the lungs, since long delay makes it more difficult to expel them and the condition of the lungs is thence more gravely injured. Secondly; if, however, severe and spasmodic pains are excited by the laceration of the lungs caused by motion of prickly calculi of this kind, copious hemoptysis is to be expected; and therefore instead of lubricating medicines laudanum should be given freely and frequently, by which the motion of the calculus can be stopped promptly, pain can be relieved and hemoptysis can be checked. . . . Certainly if phthysical patients of this kind take care of their health, as by always breathing pure country air, avoid taking cold, drinking bouts, excessive exercise and whatever else can excite the fixed calculi to new movement, they will be able to live to an advanced old age, performing their usual duties satisfactorily. . . . Only when there is urgent need should a milk diet be ordered in phthisis arising from pulmonary calculi, because such a diet is likely to generate new chalky stones of this kind in the lungs. Whence it happens that the cure of one phthisis is accustomed to lay the foundation for another.

He reported 2 cases of broncholithiasis.

Boerhaave,⁴ in 1744, described the case of the botanist, Sebastian Vaillant, who, through many years, coughed up 400 broncholiths and died at 54 years of age of "phthisis calculeuse."

Morgagni¹ stated that "almost 100 authors have described calculi, or tophi, that have been brought up by coughing, or found in the lung." He did not indicate how many of these were bronchial calculi. He again brought up the subject as to whether milk should be used in patients who had broncholithiasis. He agreed with Morton, feeling that it was contraindicated. He quoted other authors, among them an Albertus Fabrius, who contended "very resolutely" that calcareous concretions in the lung might be increased by the use of milk. He cited the case of a patient who craved milk "to such an extent that he drank it even instead of water or beer." It was felt that this program

Read at the Twenty-ninth annual meeting of The American Association for Thoracic Surgery, New Orleans, La., March 29-31, 1949.

*Division of Medicine, Mayo Clinic.

†Division of Surgery, Mayo Clinic.

‡Division of Surgical Pathology, Mayo Clinic.

It might be well to mention metastatic calcification of the lung. Best and Taylor stated that this is a condition wherein there is a translocation of the calcium of the skeleton to the soft tissues. It occurs when excessive doses of parathyroid extract or irradiated ergosterol have been used. The calcareous deposits may occur in any of the soft tissues. However, the kidneys, arteries, lungs, and the fundus of the stomach are the most common parts that are involved. The kidneys, the stomach, and the lungs are organs that eliminate acid, and theoretically, at least, the cells of these organs might be left more alkaline in reaction. A situation favorable for the deposition of calcium is created. Metastatic calcification may occur in hyperparathyroidism, renal rickets, diseases of bone such as multiple myeloma and osteomalacia, and malignant bone tumors.

It is possible that the phenomenon of metastatic calcification is due to excess calcium in the blood. It has been suggested¹⁹ that, in some instances, such calcification is secondary to tissue injury and therefore is, in reality, a type of dystrophic calcification induced by a toxic agent. It has been shown that parathyroid extract and irradiated ergosterol have a definite toxic reaction other than their effect on calcium metabolism. In some instances, metastatic calcification is associated with hypocalcemia.

Wells²⁰ said that when metastatic calcification accompanies destructive disease of bone, calcium salts are absorbed in greater amounts than can be held in solution by the arterial blood. Under these circumstances no calcium is found to be deposited on the right side of the heart. After the blood has passed through the lungs and has lost a large part of its carbon dioxide, the calcium salts may be found precipitated in the pulmonary veins and the left side of the heart. The lungs are also a site of calcium deposition under these circumstances. No cellular action has been demonstrated to account for the deposition of these calcium salts. It has been felt that the deposition of calcium salts in tracheobronchial lymph nodes is probably due to a process of filtration alone. We have not observed an instance of broncholithiasis which has occurred secondary to metastatic calcification.

Pathologic Aspects.—Poulalion²¹ was one of the first to describe the pathologic features of broncholithiasis. He stated that broncholiths may be cartilaginous, calcareous, or osseous in their make-up and that they may arise anywhere in the pleuropulmonary tissues. Pendergrass and de Lorinier,²² in attempting to explain the pathogenesis of pulmonary calcification, postulated four possibilities: (1) secondary calcification after degeneration or necrosis due to infection, (2) primary or secondary calcification of aspirated foreign bodies, (3) precipitation of the osseous inorganic elements due to excessive concentrations of them in the blood, and (4) metastasis of bone-forming cells.

It has been said that broncholiths may originate (1) in the lumen of the bronchus, (2) in the bronchial wall, or (3) around the bronchus and subsequently erode through the wall of the bronchus. It has been stated that aspirated foreign bodies and endobronchial secretion may act as nuclei for the deposition of calcium.¹¹ Inflammatory processes in the tracheobronchial

tree may lead to necrosis of the tracheobronchial cartilages which likewise may act as nuclei in the formation of broncholiths.

Most workers feel that the great majority of broncholiths arise when tracheobronchial lymph nodes become calcified secondary to an antecedent pulmonary infection. These calcified lymph nodes erode the bronchial wall and gain partial or complete entrance into the bronchial lumen. Such a broncholith acts like a foreign body.

Broncholiths may vary in size from granules to the size of 139 Gm.⁹ They usually are gray-white, irregular, and hard, but they may look like putty. They usually are rough and irregular. In most instances, when either coughed up or removed bronchoscopically, they are broken so that they may represent merely a fragment of a large stone. They may show black areas of anthracosis. A broncholith which is not coughed up promptly is quite likely to cause collapse of the lung distal to the site of obstruction. Subsequently, pneumonitis, bronchiectasis, lung abscess, or empyema may follow owing to bronchial obstruction and secondary infection.

Fox and Clerf,²³ using iodized oil, were able to demonstrate cylindrical bronchiectatic lesions distal to the site of obstruction in 4 cases after removal of the broncholith.

DATA ON FORTY-ONE MAYO CLINIC CASES

Cases Included.—Included in this series are the cases in which there was a history that one or more broncholiths had been coughed up but in which, at the time of examination, no broncholith could be found. Included also, of course, were the cases in which a broncholith was found either free or fixed in the lumen of the bronchus at the time of bronchoscopy or surgical treatment. At least a part of the broncholith must have eroded into the lumen of the bronchus before the case was eligible for inclusion. The distinction, we feel, is important since calcified lymph nodes that create only extrinsic pressure on a bronchus should not be designated as broncholiths.

Since the publication of the article by Tinney and Moersch in August, 1944, which was based on 28 cases, an additional 13 cases of broncholithiasis have been encountered at the clinic. It is of interest to note that broncholithiasis was recognized in only 28 patients in the thirty-seven years which preceded August, 1944, but that it has been recognized in 13 patients in the succeeding four years. This rapid increase in incidence has occurred by virtue of the fact that an increasing number of indeterminate pulmonary lesions are being explored surgically at an early date to make certain that they are not bronchiogenic carcinomas. In 8 of the cases of broncholithiasis encountered since 1944, the condition was diagnosed at the time of surgical exploration, and in only 3 was the lesion recognized at the time of bronchoscopy. Exploration carried out in 2 additional patients showed pathologic changes secondary to the presence of broncholiths which had been expectorated prior to operation.

This review covers the entire series of 41 cases encountered at the clinic, including the 28 cases reported by Tinney and Moersch. By basing our analysis on the entire series of cases, we believe that the validity of the statistical data obtained is improved.

Twenty-one of the patients were women and 20 were men. The age distribution was as follows: 20 to 29 years, 1 case; 30 to 39 years, 6 cases; 40 to 49 years, 18 cases; 50 to 59 years, 11 cases; 60 to 69 years, 3 cases, and 70 years or more, 2 cases. Thus, 71 per cent of the cases occurred in the age group of 40 to 59 years.

The diagnoses in these cases previous to examination at the clinic were as follows: tuberculosis, 9 cases; carcinoma, 4; bronchial obstruction with stenosis, 4; bronchiectasis, 3; pneumonia, 2; broncholithiasis, 2; lung abscess, 1; and empyema, 1. No previous diagnosis had been made in the remaining 15 cases.

Symptoms.—Broncholiths are foreign bodies in the tracheobronchial tree and thus are usually very irritating. The severity of symptoms depends on the size and location of the broncholith. The symptoms may vary in degree from an unconscious raising of a small stone with a mild cough to the severe stone asthma or bronchial colic with which there may be intense substernal pain accompanied by a sense of suffocation.

The duration of symptoms in this group of patients was of interest. According to the history, the shortest duration was thirty days, and the longest, twenty-two years. The first symptom was cough in 23 cases (56 per cent), hemoptysis in 9 cases (22 per cent), and thoracic pain in 9 cases (22 per cent).

The most common symptom was cough, which occurred in all cases. The cough was productive of only stony material in 1 case. In 25 cases (61 per cent) there was slight to moderate expectoration. Eight patients (20 per cent) gave a history of coughing up as much as 2 ounces (about 60 c.c.) of mucopurulent secretion in twenty-four hours. Seven (17 per cent) raised from 3 ounces (about 90 c.c.) to more than one cupful (about 240 c.c.) of mucopurulent secretion in twenty-four hours.

The next most frequent symptom was hemoptysis. This occurred in 35 cases (85 per cent). Six of these patients had undergone such severe hemorrhage that they had required transfusion.

Fever, usually in association with chills, had occurred in 25 cases (61 per cent). Pleuritic pain occurred in 23 cases (56 per cent), and at times this pain was severe. The pain may occur in the retrosternal region and extend to the thoracic spinal region. A certain number of patients had an asthmatoïd wheeze. At times this was severe, resulting in intractable asthma.

Physical Findings.—The physical findings were not particularly diagnostic in the great majority of the patients. The physical findings depended on the size of the broncholith and its location. If a broncholith was located in a tertiary bronchus, physical findings were sometimes absent. If the broncholith was located in a secondary bronchus and caused a high degree of bronchial obstruction, the physical findings were those seen in collapse of a lobe. Thus, there would be dullness to percussion and decreased or absent breath sounds over the region affected. The mediastinum was displaced toward the affected side and the intercostal spaces were decreased in size. Sometimes there was an inspiratory lag on the affected side and elevation of the diaphragm. One of the earliest findings was usually the reduction of the intensity of breath sounds

over the affected part. Sometimes a wheeze was heard over the region; at times this wheeze was heard only at the end of a deep inspiration.

Roentgenologic Findings.—The roentgenologist suspected broncholithiasis in only 3 of the 41 cases. The roentgenologic diagnoses were as follows: obstructing lesion with atelectasis, 6 cases; bronchiectasis, 4; calcified hilar nodes, 7; "rule out bronchiogenic carcinoma," 6; pulmonary abscess, 5; broncholithiasis, 3; tuberculosis, 3; negative for thoracic disease, 2; infiltration of hilus, 1; multiple areas of pulmonary calcification, 3; and right pneumothorax and fibrosis of both upper lobes, 1. In the last-mentioned case, the patient had had a right artificial pneumothorax induced prior to his arrival at the clinic, in an attempt to control pulmonary hemorrhage.

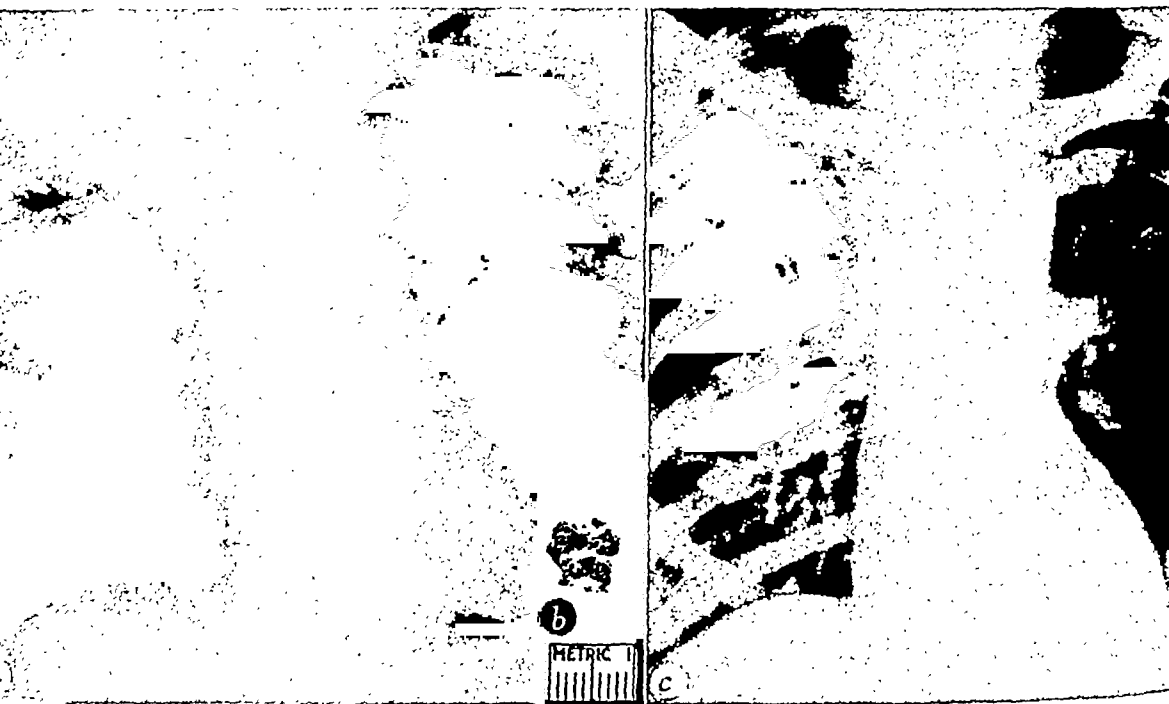


Fig. 1.—a. Broncholith occluding right lobe bronchus. The broncholith could be felt with grasping forceps at the time of bronchoscopy but could not be removed. The right upper lobe bronchus was dilated. b. The stone that was expectorated immediately after bronchoscopy. c. Appearance one month after bronchoscopy. (From Tinney, W. S. and Moersch, H. J.: Broncholithiasis, *S. Clin. North America* 24: 830-838, 1944.)

In retrospect, probably the most diagnostic roentgenologic finding, when it occurred, was the presence of a dense area of calcium deposition at the apex of a triangular portion of collapsed lung (Fig. 1). The apex of the triangular shadow was located at the mediastinum, and the portion of the lung that was collapsed corresponded to a segment or a lobe. At times this area of calcification was hidden by the heart, and heavy-exposure technique was required to visualize it.

With the collapse of all or a portion of the lung, there may be diminution in the size of the affected pulmonary field. Thus, the diaphragm may be elevated, the mediastinum shifted to the affected side, and the intercostal spaces decreased on this side.

Broncholithiasis may cause pulmonary abscess or empyema, and thus the roentgen findings may be those of these complications. If the broncholith is small, the roentgenologic examination of the thorax may give negative results (Figs. 2 and 3).

Some roentgenograms showed only multiple areas of calcification throughout both lungs. Others revealed the picture of unilateral or bilateral hilar calcification without any evidence of collapsed lung.

Bacteriologic Observations.—The sputum was examined for tubercle bacilli by direct smear in all cases and was found to be free of such microorganisms.

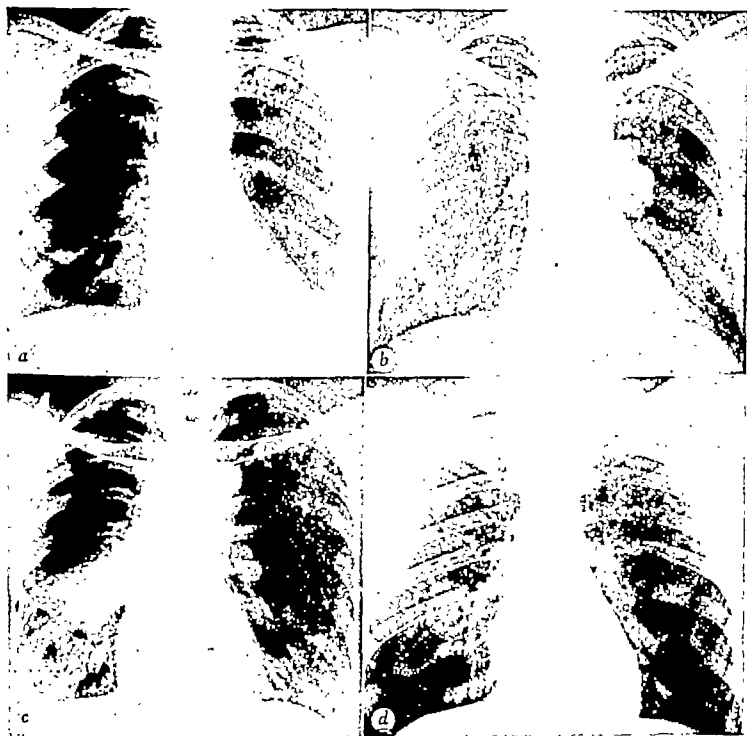


Fig. 2.—Variation in roentgenographic appearance in broncholithiasis. *a*, The appearance is essentially normal yet the bronchoscope found and removed a broncholith from the left main bronchus. *b*, Extensive calcification of the tracheobronchial lymph nodes. A broncholith was found and removed from the left lower lobe by the bronchoscope. *c*, Broncholitis may produce the roentgenographic findings of pulmonary abscess. In this case, a right lower lobectomy was performed. Two broncholitis were found in the depths of the right lower lobe bronchus. Active pulmonary tuberculosis was also found. Guinea pigs inoculated with material taken from the right lower lobe were found to be positive for tuberculosis. *d*, The lesion may resemble those seen in coccidioidomycosis, with a well-circumscribed annular shadow in which there is a central area of rarefaction.

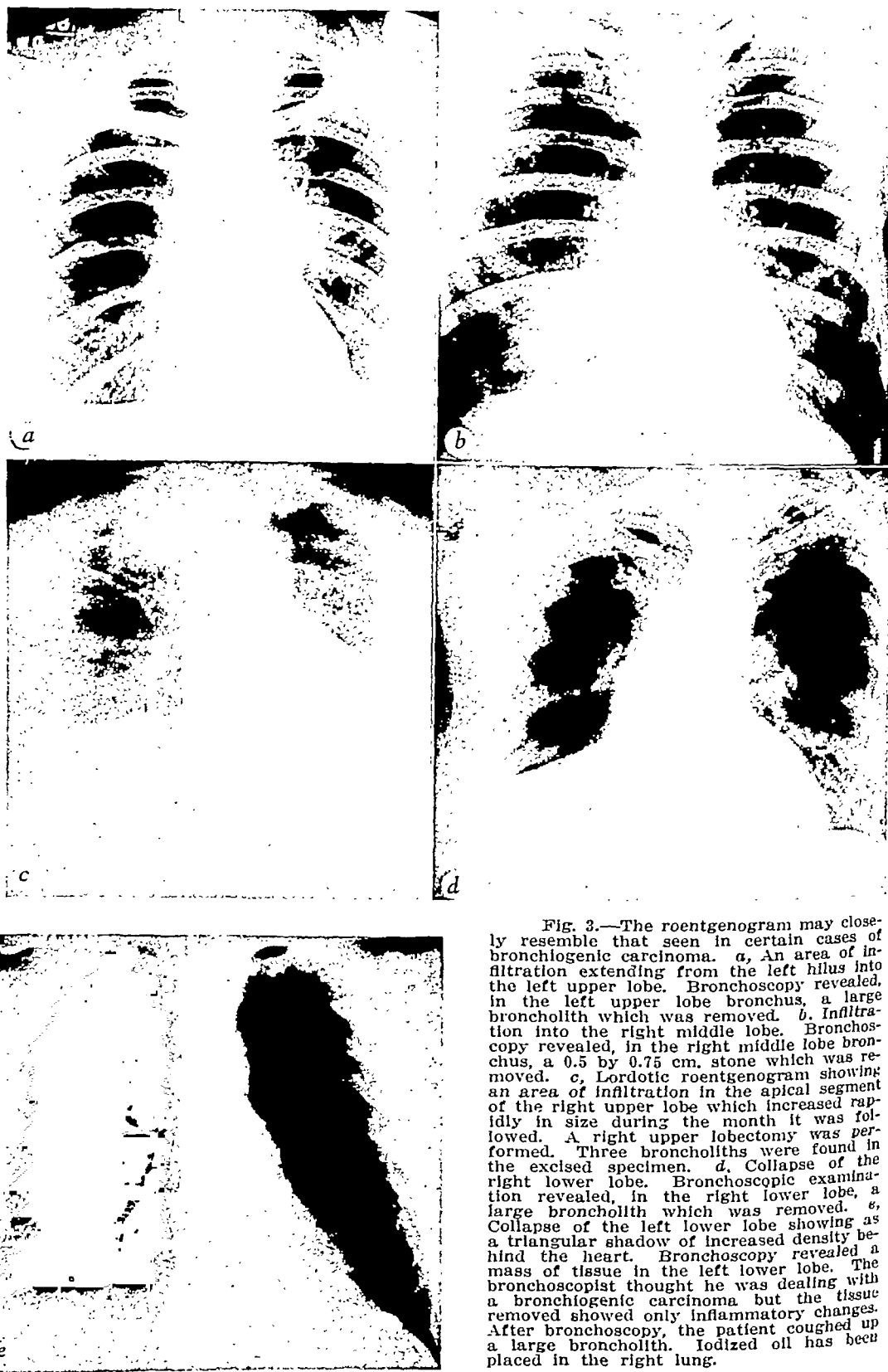


Fig. 3.—The roentgenogram may closely resemble that seen in certain cases of bronchiogenic carcinoma. *a*, An area of infiltration extending from the left hilus into the left upper lobe. Bronchoscopy revealed, in the left upper lobe bronchus, a large broncholith which was removed. *b*, Infiltration into the right middle lobe. Bronchoscopy revealed, in the right middle lobe bronchus, a 0.5 by 0.75 cm. stone which was removed. *c*, Lordotic roentgenogram showing an area of infiltration in the apical segment of the right upper lobe which increased rapidly in size during the month it was followed. A right upper lobectomy was performed. Three broncholiths were found in the excised specimen. *d*, Collapse of the right lower lobe. Bronchoscopic examination revealed, in the right lower lobe, a large broncholith which was removed. *e*, Collapse of the left lower lobe showing as a triangular shadow of increased density behind the heart. Bronchoscopic examination revealed a mass of tissue in the left lower lobe. The bronchoscopist thought he was dealing with a bronchiogenic carcinoma but the tissue removed showed only inflammatory changes. After bronchoscopy, the patient coughed up a large broncholith. Iodized oil has been placed in the right lung.

In some instances, cultures for tubercle bacilli and fungi were performed with negative results except in 1 instance. In only 1 of the surgical cases was evidence of active tuberculosis found. In this case, guinea pigs inoculated with material from the resected lung were found to be positive for tuberculosis.

Bronchoscopic Data.—After the history, physical examination, and roentgen studies of the thorax and bacteriologic studies of the sputum have been completed, one may or may not think of the possibility of broncholithiasis. Usually, in our series of cases, the clinician considered bronchiogenic carcinoma as being the most likely diagnosis prior to bronchoscopy.

Six of the 41 patients did not undergo bronchoscopy. These were persons who gave a history of having coughed up stones prior to coming to the clinic but who, at the time they were seen, were asymptomatic. Thirty-five patients underwent bronchoscopy one or more times; 32 (91 per cent) of these had positive findings; in 17 instances (48 per cent) the broncholith was either found and removed or coughed up immediately after bronchoscopy. In 1 additional case the broncholith was found but could not be removed. In 10 cases the involved bronchus was found to be strictured or obstructed by granulation tissue.

The bronchoscopic findings often were at first suggestive of a tumor since frequently the broncholith was buried in a fleshy mass of granulation tissue. When this tissue was removed by biopsy forceps, the broncholith was felt or seen. It is extremely important to do a biopsy on this tissue since broncholiths may occur in association with bronchiogenic carcinoma. The surrounding mucous membrane was inflamed, and often a large amount of mucopurulent secretion was dammed up distal to the stone. In 4 cases, bloody secretion was found to be coming from the involved bronchus. In 3 instances bronchoscopy gave negative results.

Once the broncholith is removed, the bronchial mucous membrane usually returns to normal. There is usually no residual bronchial stricture. If bronchiectasis develops distal to the broncholith, the bronchoscopist will see purulent secretion coming from that portion of the bronchial tree distal to the site of the broncholith.

The bronchoscopic examination may not reveal the diagnosis, depending on the location of the stone. If the stone can be seen, it usually can be removed. If it is deeply embedded in an upper lobe bronchus, it may be impossible to grasp it securely enough to remove it. By dilating the bronchus, the broncholith may be coughed up after bronchoscopy.

Two complications which may occur following bronchoscopy should be mentioned. They are (1) hemorrhage and (2) pneumothorax on the involved side.²⁴ They are not usually serious complications.

Surgical Treatment.—If the broncholith is located in a part of the bronchial tree that cannot be visualized by the bronchoscopist, the problem of a peripherally placed indeterminate bronchial lesion confronts the physician. In our experience it is far safest to explore such a lesion early since a large percentage of these lesions have turned out to be bronchiogenic carcinomas. At the time of exploration the broncholith will be found, and segmental resection, lobectomy, or pneumonectomy may be necessary.

In the future, more and more bronchololiths will be found by the thoracic surgeon at the time of thoracotomy. Myers,²⁵ in reporting 4 cases of broncholithiasis, stated that in 1 of the cases the diagnosis was made when a right lower lobectomy was performed for intractable hemorrhage from this lobe. VanOrdstrand and co-workers,¹⁰ in reporting 2 cases, performed right pneumonectomy for an indeterminate lesion. Before and at the time of the operation, it was believed that a bronchiogenic carcinoma might be present. A calcified nodule was found to be obstructing the bronchus of the right middle lobe by exerting extrinsic pressure. Anderson and MacKay²⁶ reported a case of an indeterminate lesion involving the right middle lobe. This lesion caused a collapse of the lobe. The bronchoscopist did not make the diagnosis at the time of his examination. A right middle and right lower lobectomy was performed. At this time, 3 calculi were found in the right middle lobe. The calculi had originated in calcified lymph nodes that lay posterior to the bronchus and had eroded through its wall.

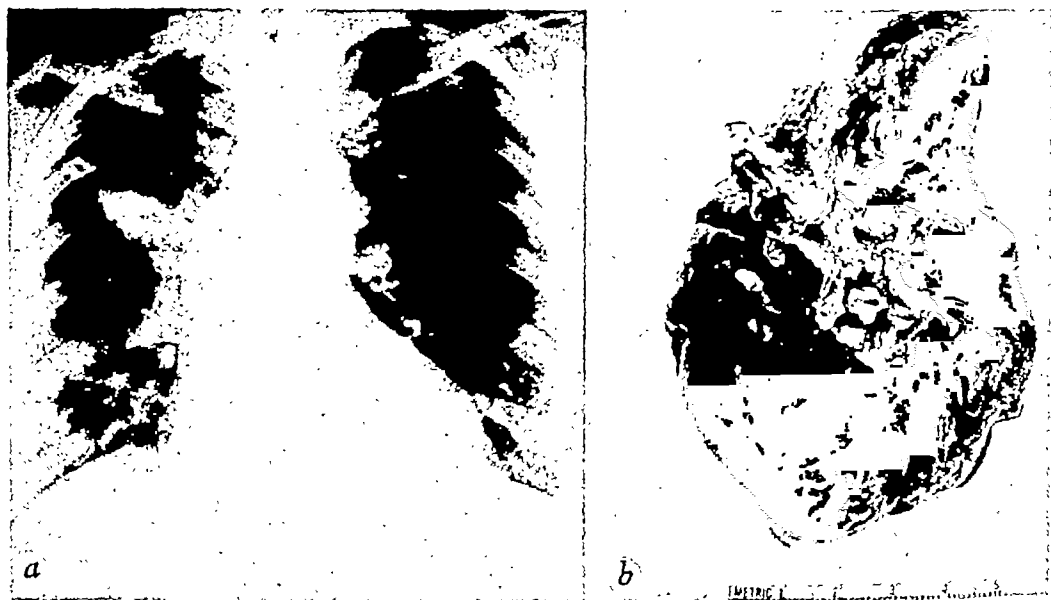


Fig. 4.—*a*, Area of obstructive pneumonitis in the anterior segment of the right upper lobe shown in roentgenographic study. A right upper lobectomy was performed, which revealed an irregular, whitish bronchololith in the upper lobe bronchus with marked bronchiectasis distal to the bronchololith. *b*, The bronchololith.

There were 10 cases in our series in which it was necessary to remove part or all of the lung. Nine of these represented removal of a portion or all of the right lung, and only 1 represented removal of a portion of the left lung. In 2 of these cases the bronchololith had been coughed up a short time prior to operation and no bronchololith was demonstrated at operation. In these 2 cases, a residual stricture was present which necessitated lobectomy. In both cases, there was marked obstructive pneumonitis in the periphery of the affected lobe. The location of the bronchololith in the remaining 8 cases was as follows: 3 were in the upper lobe, 1 was in the middle lobe, 2 were in the main stem bronchus on the right side, and 2 were in the lower lobe bronchus. In 3 instances there was more than 1 bronchololith, while in 5 there was only 1 (Fig. 4).

The largest broncholith measured 1.5 cm. in diameter, the smallest 0.3 cm. The broncholiths were more frequently whitish than any other color. A few were yellowish, and others were black, indicating the deposition of carbon pigment within their substance. Broncholiths usually have irregular knobs on them and look much like a staghorn calculus of the kidney in miniature.

There were no surgical deaths in our series. Empyema developed in 1 patient postoperatively, which necessitated performing external drainage. He made an uneventful recovery after this operation.

Nineteen of our patients gave a history of having coughed up multiple broncholiths nonsynchronously. One patient stated that he had coughed up 103 stones. Because of the possibility of multiple sites of origin, we feel that any surgical procedure directed toward the eradication of this type of lesion should be as conservative as possible to preserve the maximal amount of pulmonary function.

Pathologic and Etiologic Features.—Histologic examination of the broncholiths revealed no structure because of cellular death as the result of detachment from their blood supply. The general conclusion reached from the chemical examination of 3 broncholiths in this series is that the chemical constituents are about right for bone (Table I).

TABLE I. COMPOSITION OF BRONCHOLITHS

CASE	WET SAMPLE (MG.)	DRY SAMPLE (MG.)	ASH (MG.)	CALCIUM IN SAMPLE (MG.)	CALCIUM IN ASH (PER CENT)	PHOSPHORUS IN SAMPLE (MG.)	PHOSPHORUS IN ASH (PER CENT)	CALCIUM- PHOSPHORUS RATIO	VOLATILE MATTER (PER CENT)
1	99.1	97.3	83.8	30.5	36.4	14.0	16.7	2.18:1	13.9
2	90.0	93.3	79.0	28.7	36.1	13.3	16.7	2.16:1	14.7
3	106.9	100.3	81.4	28.1	34.5	12.8	15.8	2.2:1	19.1

The causes of calcification in the lung and bronchial lymph nodes are varied. The granulomatous diseases may produce calcification or ossification in the lung and the hilar lymph nodes. The most frequent of these diseases is tuberculosis. Calcification or ossification is common in the Ghon complex, either in the periphery of the lung or the hilar nodes (Fig. 5), but may be seen also in the adult form of tuberculosis. It seems likely to us that the peripheral calcification from any cause is of little importance in the production of broncholiths because it is unrelated to a bronchus which is large enough to permit the nodule to ulcerate into its lumen. On the other hand, it would appear that calcareous or ossified peribronchial lymph nodes, in particular those associated with larger bronchi, are capable of ulcerating into a bronchus and eventually becoming detached. Of the other granulomatous diseases which produce calcification, the most noteworthy is coccidioidomycosis and histoplasmosis.

A second type of calcification (Fig. 6a) or ossification (Fig. 6b) in the lung is that seen in the elastic cartilage of the bronchi. Cartilage is seen in the wall of bronchi larger than 1 mm. The process of calcification and ossification of the cartilage is unrelated to inflammation but is, rather, a part of the picture of senescence. It is very commonly seen, particularly in older persons. Such calcareous or ossified plaques, when formed in the wall of bronchi, would



Fig. 5.—Calcification of a lymph node subjacent to the main bronchus (hematoxylin and eosin, $\times 9$).

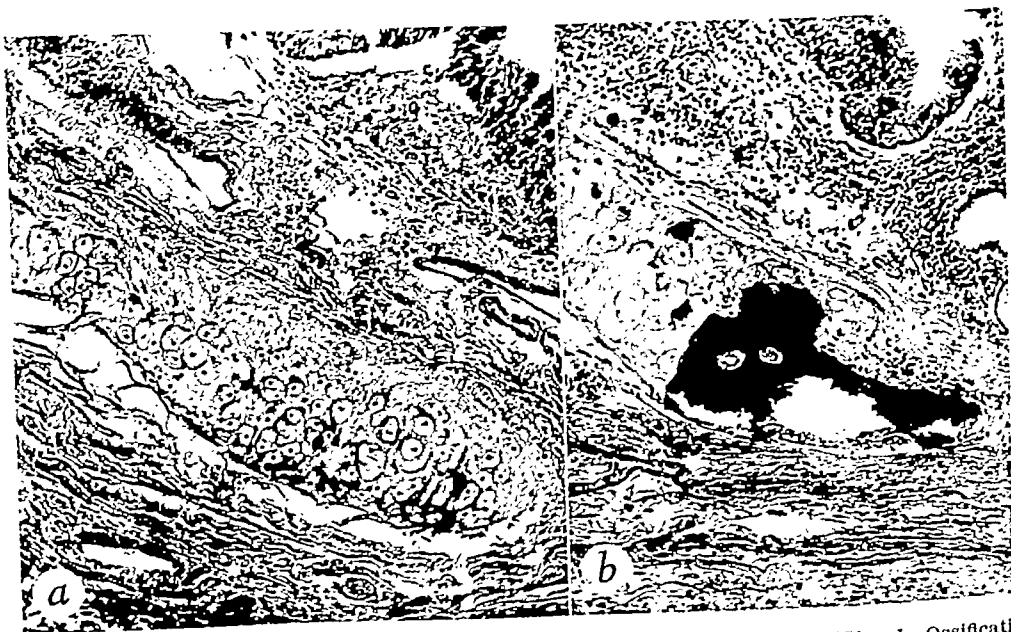


Fig. 6.—*a*, Calcification of bronchial cartilage (hematoxylin and eosin, $\times 105$). *b*, Ossification of bronchial cartilage (hematoxylin and eosin, $\times 95$).

be just as capable of sequestering into the bronchial lumen as would lymph nodes which had undergone similar changes. As a matter of fact, such plaques in cartilage are originally in the wall of the bronchus while those in lymph nodes are situated outside the bronchial wall. Therefore, those in cartilage would appear to form broncholiths more readily by sequestration because of their proximity to the bronchial lumen than would calcified lymph nodes.

The third type of calcification or ossification is that seen in metastatic calcification. This is the condition in which "calcium salts are deposited in tissues previously normal but with a tendency to alkalinity." Metastatic calcification of the lungs is seen in osseous disease, renal disease, hypervitaminosis D, and hyperparathyroidism. The calcium is deposited usually in the periphery of the lung, in the walls of blood vessels, and in the walls of bronchi. It seems very unlikely that this type of calcification leads to broncholithiasis very often, inasmuch as it is found in the peripheral part of the lung, and such certainly has not been the case in our experience.

Calcification or ossification is also seen in the lung in metastatic sarcoma, particularly osteogenic sarcoma and chondrosarcoma. In addition, hamartomas frequently show calcification or ossification. It is obvious that this bears little relation to the problem of broncholithiasis. Our experience would indicate that the first two processes, namely, calcareous or osseous change in peribronchial lymph nodes subjacent to larger bronchi, as well as calcareous or osseous changes in bronchial cartilages, are the two most important factors in the production of broncholithiasis. Of the 10 cases in which the lung was available for study, ossified or calcareous cartilages were present in 9 cases, ossified or calcareous nodes were present in 5 cases, and in 1 case there was no calcific change in either the bronchial cartilage or the bronchial lymph nodes which were removed with the specimen. It is impossible, of course, in a given case, to be sure which of these two factors played the important role, although in the 5 cases in which calcareous or ossified nodes were present, it seemed to us very likely that the source of the broncholith was a lymph node. In addition, in the 2 cases in which the broncholiths were black, this also would indicate an origin from lymph nodes. The calcified or ossified lymph node or bronchial cartilage appears to act as a foreign body, and it extrudes through the bronchial wall into the lumen. The mucosa of the bronchus at the site of the broncholith is raised into folds and there is subjacent inflammation and granulation tissue (Figs. 7 and 8); as a result, the broncholith is enmeshed so that oftentimes it became invisible on cursory gross examination. It is only after exploring the narrowing of the bronchus that one locates the underlying trouble. As has been seen in 2 cases in which surgical resection was performed even after removal of the broncholith, a permanent stricture may result. The periphery of the lung in all cases showed the condition which has been called "obstructive pneumonitis." In this condition the bronchi are usually dilated, the pulmonary alveoli and bronchi are filled with exudate, and there are varying degrees of fibrosis dependent upon the time during which the obstruction has been operative. This pneumonitis is no different in obstruction caused by broncholithiasis from what it is in any other type of obstruction, and it is for this reason that broncholithiasis simulates carcinoma of the lung.

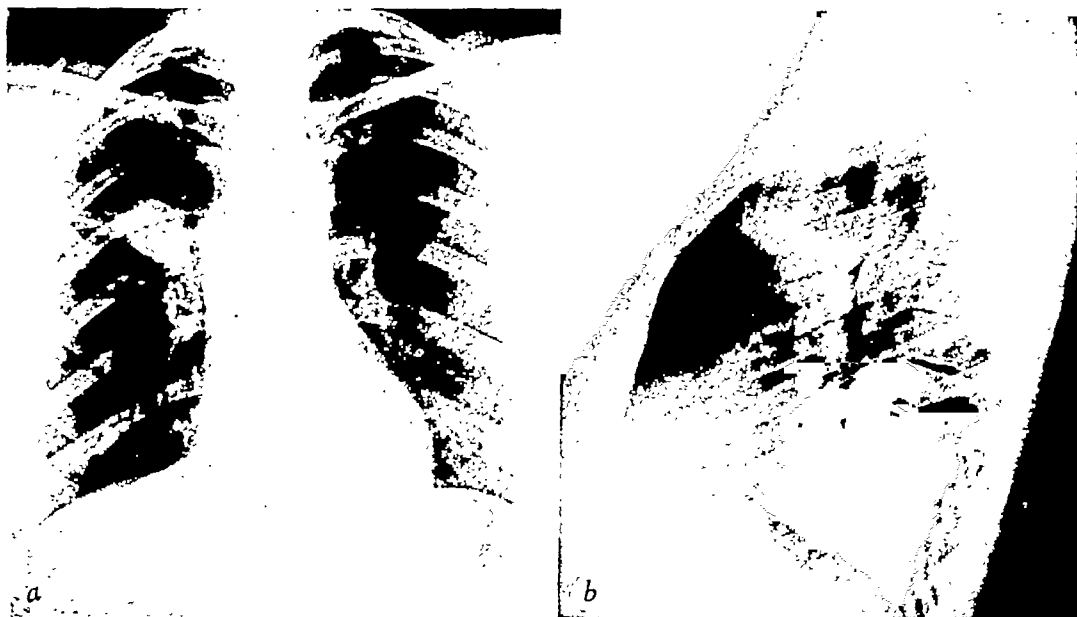


Fig. 7.—Lesions in the anterior segment of the right upper lobe. Bronchoscopy revealed a stenosed bronchus of the right upper lobe. A right upper lobectomy was done. There were multiple broncholiths in the right upper lobe bronchus. *a*, Anteroposterior view, *b*, lateral view.



Fig. 8 (Same case in Fig. 7).—Bronchial wall subjacent to a broncholith. Note the inflammatory reaction in the mucosa of the bronchus (hematoxylin and eosin, $\times 95$).

Active tuberculosis was not a feature in the pulmonary tissue under consideration. In only 1 case was active tuberculosis found in the lobe. In this patient the results of guinea pig inoculation were positive. In 1 other case, there were giant cells and a granulomatous reaction around the ossified peribronchial lymph nodes suggestive of activity.

The location of the broncholith could be identified accurately from the records in 28 cases. In 3, the right main bronchus was involved; in 2, the right-stem bronchus contained the broncholith. The location of the rest of the stones was as follows: right upper lobe, 6 cases; right middle lobe, 3; right lower lobe, 6; left main bronchus, 1; left upper lobe, 2; and left lower lobe, 5. The right lung was involved twenty times and the left lung eight times.

If most broncholiths originate outside the bronchial tree in calcified lymph nodes, as we believe they do, one could partly explain the location of the stones by the fact that there are a greater number of lymph nodes associated with the right lung than with the left. There are a greater number of lymph nodes which lie in association with the right upper lobe bronchus than with those of the right middle or lower lobe. Miller²⁷ stated that lymphoid tissue in the lung increases from childhood to old age. He said that this increase is due largely to the irritation that arises from the constant inhalation of irritating substances.

Follow-up.—In the group of 13 cases in which either bronchoscopy was performed and no evidence of broncholithiasis was seen or in which bronchoscopy was not done because it was not thought to be indicated, 1 patient was in excellent health on follow-up study. One had had no further trouble so far as the lungs were concerned, but died of coronary occlusion at the age of 63 years, twelve years after his visit to the clinic. Four patients raised additional broncholiths after leaving; 1 stated that he had raised 40 stones after his visit, but other than this he felt that he was in good general health. Two patients continued to raise bloody sputum. In 5 instances accurate follow-up information could not be obtained.

The 18 patients who were found to have broncholithiasis at the time of bronchoscopy had the following results: Eleven became asymptomatic after the removal of the broncholith. One patient regarded herself as being in excellent health "except I still spit up" thirteen years after removal of the broncholith. One patient, 64 years of age, stated that he was in excellent health except for "some shortness of breath on exertion" seven and one-half years after removal of a broncholith. One woman, aged 70 years, who had, firmly embedded in the right middle lobe bronchus, a broncholith which could not be removed, stated that she was asymptomatic on our survey. Three patients in this group had poor results. One man had a good immediate result after removal of the broncholith, but his widow recently wrote that "his lung condition gradually grew worse ending in a heart attack and a hemorrhage." Death occurred in this case ten years after the broncholith had been removed. One patient had a contracted left lower lobe which did not expand after a broncholith was coughed up after bronchoscopy. A left lower lobectomy was

advised but the patient refused to have the operation. A lung abscess developed in 1 patient, with a metastatic abscess of the brain following bronchoscopy; he subsequently died.

Eight of the 10 patients who underwent surgical resection for broncholithiasis had excellent results. They have had no further symptoms referable to their lungs. In 1 patient, there developed an empyema which necessitated external drainage. One patient stated that she had "cardiac asthma" and "fluid" on the remaining lung three years after a right pneumonectomy. It was felt that these symptoms were due to inadequate pulmonary function.

SUMMARY

Some of the historical features of broncholithiasis are recorded. Data from the standpoint of its incidence, etiology, and clinical findings are given.

A review of data on 41 patients who were seen at the Mayo Clinic because of this condition is presented. Twenty-one of the patients were women and 20 were men. Seventy-one per cent of the cases occurred in the age group of 40 to 59 years. In 15 cases the diagnosis was made on the basis of a history that the patient had coughed up a broncholith. In 18 cases the diagnosis was made at the time of bronchoscopy, and in 8 it was made at the time of operation. The last-mentioned group of patients underwent exploration because bronchiogenic carcinoma could not be ruled out prior to operation.

Because of the possibility of multiple sites of origin, it is felt that the surgical procedure directed toward the eradication of this type of lesion should be as conservative as possible in order to preserve the maximal amount of pulmonary function.

REFERENCES

1. Morgagni, J. B.: *The Seats and Causes of Diseases*, Vol. 1, (translated by Benjamin Alexander), London, 1769, A. Millar & T. Cadell, p. 371.
2. Schenck a Grafenberg, Joannes: *Observationum medicarum, rararum, novarum, admirabilium, et monstrosarum tomus unus [et alter] in quibus quae medici doctissimi et exercitatissimi addita, vulgo incognita, gravia, periculosaeque, in harum partium conformationibus, eorundemque morborum caussis, signis, eventibus, et curationibus, accidere compererunt, exemplis ut plurimum et historiis proposita exhibentur*, Vol. 1, Frankfurt, 1600, J. Rhodii, pp. 351-355.
3. Morton, Richard: *Phthisiologia, seu exercitationes de phthisi tribus libris comprehensae. Totumque opus variis historiis illustratum*, Chap. 6, London, 1689, S. Smith, pp. 266-280.
4. Boerhaave: Quoted by Legry,⁶ p. 337.
5. Bayle, G. L.: *Recherches sur la phthisie pulmonaire*, Paris, 1810, Gabon, pp. 33-34.
6. Legry, T.: *Les pierres du pouton*, Arch. gén. d. méd. 169: 337-349, 466-475, 1892.
7. Stivelman, B. P.: *Broncholithiasis; Report of a Case*, Am. Rev. Tuberc. 18: 430-434, 1928.
8. Pritchard, J. S.: *Some Interesting Cases of Calcareous Degeneration Found in the Thorax*, Arch. Int. Med. 32: 259-282, 1923.
9. Lloyd, J. J.: *Broncholiths. With Report of Four Cases*, Am. J. M. Sc. 179: 694-699, 1930.
10. VanOrdstrand, H. S., Moore, P. M., Jr., and Harris, H. E.: *Broncholithiasis; Report of Two Cases*, Cleveland Clin. Quart. 9: 36-44, 1942.
11. Tinney, W. S., and Moersch, H. J.: *Broncholithiasis*, S. Clin. North America 24: 830-838, 1944.
12. Sternberg, Carl: *Ueber die Erweichung bronchialer Lymphdrüsen und ihre Folgen*, Wien. klin. Wchnschr. 18: 1214-1215, 1905.
13. Zahn, D. W.: *Broncholithiasis*, Am. Rev. Tuberc. 54: 418-423, 1946.

14. Lell, W. A.: Broncholithiasis; Report of Six Cases, *Tr. Am. Broncho-Esophagol. A.* pp. 130-140, 1917.
15. Best, C. H., and Taylor, N. R.: *The Physiological Basis of Medical Practice; a University of Toronto Text in Applied Physiology*, Baltimore, 1943, The Williams & Wilkins Company, 1942 pp.
16. Wells, H. G.: Calcification and Ossification, *Arch. Int. Med.* 7: 721-753, 1911.
17. McPhedran, Alexander: Diseases of the Bronchi: Bronchial Calculi (Broncholiths), in Osler, William: *Modern Medicine; Its Theory and Practice*, Vol. 4, Chap. 5, Philadelphia, 1927, Lea & Febiger, pp. 147-148.
18. Bech, Knud: Broncholithiasis, *Nord. med.* 30: 810-812, 1946.
19. Feldman, W. H., and Baggenstoss, A. H.: The Residual Infectivity of the Primary Complex of Tuberculosis, *Am. J. Path.* 14: 473-490, 1938.
20. Wells, H. G.: Metastatic Calcification, *Arch. Int. Med.* 15: 574-580, 1915.
21. Poulalion, S. A. M.: Les pierres du poumon, de la plèvre et des bronches, et la pseudo-phthisie pulmonaire d'origine calculeuse, Thesis, Paris, 1891, G. Steinheil, 240 pp.
22. Pendergrass, E. P., and de Lorimier, A. A.: Broncholiths and Stone Asthma, *Radiology* 25: 717-722, 1935.
23. Fox, J. R., and Clerf, L. H.: Broncholithiasis: Report of Ten Cases, *Ann. Int. Med.* 23: 955-959, 1945.
24. Schmidt, H. W.: Broncholithiasis, *Proc. Staff Meet., Mayo Clin.* 13: 609-612, 1938.
25. Myers, D. W.: Broncholithiasis, *Dis. Chest* 6: 269-272, 1940.
26. Anderson, W. S., and MacKay, J. B.: Broncholithiasis, *Dis. Chest* 10: 427-432, 1944.
27. Miller, W. S.: *The Lung*, Springfield, Ill., 1937, Charles C Thomas, Publisher, 200 pp.

DISCUSSION

DR. JEROME HEAD, Chicago.—I have been very interested in this subject for a number of years. Dr. Mohan and I presented a paper on the subject at the Trudeau Society meeting in New York last June, which will be published in the *American Review of Tuberculosis* in the next month or so. I think the condition is extremely common and that one will find it in proportion to the diligence with which he looks for it, as was stated in the paper. These patients frequently do say that they have coughed up stones; they think that is more or less a usual thing with a cough and they do not say anything about it. Unless one asks the patient specifically whether or not he has coughed up stones he often fails to reveal the fact. We had one patient who had had an atelectasis of a lobe; any number of doctors had examined him and questioned him, and when I made rounds I asked, "Have you ever coughed up anything peculiar?" He pulled out his pocketbook and took out an envelope with about 20 stones, which he had not shown to any other doctor—he had to be asked.

This patient had stones and a cancer; the cancer had eaten into a calcified gland and freed calcium. Most cases, of course, are caused by tuberculous calcified glands rupturing into the bronchus. We had one case recently with a typical clinical and x-ray picture of histoplasmosis in which stones were coughed up from a calcified gland in the hilus.

(Slide.) Dr. Mohan became interested in the anatomy of the glands around the roof of the lung and worked out this diagram; this has been done before and his work merely verified what has been done previously. The most important gland, I believe, is No. 4 on the right, which is the bifurcation gland; it runs down along the right-main bronchus. That is absolutely constant. No. 2, No. 1 below the upper lobe, and No. 3 are very important ones just below the middle lobe. The glands on the left side are less constant and less regular and it was pointed out that rupture into the bronchus is rather uncommon on the left side.

This is a patient in whom the gland at the bifurcation ruptured into the right main bronchus and into the esophagus. The patient had empyema and lung abscess which had broken into the pleural cavity. This was drained; Dr. Holinger used bronchoscopy and esophagoscopy, and cauterized the two openings; they healed and the patient recovered without anything but drainage of an empyema. That was many years ago and I think today the appearance of this complication would justify operation.

To show how common it is, in the last six months we have seen 6 cases, and we have under observation 3 patients at the present time in whom we feel sure that a calcified gland is causing recurrent atelectasis, and we anticipate that they will cough up calcium in the

near future. There is no set treatment for the condition. Most patients cough up a stone or two, or three or four. They have an occasional spell of fever and atelectasis, the condition will pass away, and they have no more trouble. Operation should be done only in those cases in which there is continuing and recurring trouble. A period of rest in bed and one month or so of intensive penicillin treatment will often permit the infection in the gland to clear up and the gland will heal over. Removal of the stones at bronchoscopy is relatively dangerous because of the marked tendency to hemorrhage.

DR. EDGAR W. DAVIS, Washington, D. C.—I have enjoyed immensely Dr. McDonald's presentation on broncholithiasis.

In this connection, I wish to report the case of a 50-year-old white man who had been wheezing for about two months prior to the time I saw him. A bronchoscopic examination was performed at which time some granulation tissue was found in the left-stem bronchus. A biopsy of the tissue revealed only inflammatory reaction. The following day a large stone and two small ones were coughed up. The wheezing disappeared, only to recur three months later. Another bronchoscopy was performed, revealing the same findings as on the previous examination. The symptoms of asthma disappeared and reappeared on two other occasions during the following ten months. The fourth bronchoscopic examination revealed a carcinoma in the right lower lobe bronchus, for which surgery was done. It has now been about five months since surgery and there have been no symptoms of asthma.

There is a possibility that some of these other patients with broncholithiasis may develop carcinoma.

DR. E. E. AVERY, Chicago.—I would like to show one slide of a case from Hines Hospital which demonstrates the most conservative surgical management. This 32-year-old patient had a history of recurrent pneumonitis over a period of several years. He had to stop work every two months or so and take penicillin until relieved. The bronchogram illustrates the area of stricture between the right upper and middle lobes which was seen at bronchoscopy; it was a fishmouth type with a very narrow flat orifice which could not be dilated at this time. At surgery, which was performed by Dr. Langston, two possibilities appeared; one was to resect the middle and lower lobes, but it was felt that this would remove a large amount of good functioning lung tissue. Therefore, we decided to excise the stone with its surrounding fibrous tissue and attempt to dilate the bronchus post-operatively. Bronchoscopic dilatation was then accomplished with a very good result.

This case is presented to illustrate the transthoracic excision of a broncholith with postoperative dilatation of the bronchus in an appropriate case.

DR. A. J. GRACE, London, Ont.—I should like to place one case on record. This white woman, 36 years of age, came to me in 1946, with a history of a productive winter cough for twenty years, starting when she was 16. The cough had become persistent all the year round three years before I saw her, and at that time it was more productive of mucopurulent expectoration. Soon after that she began to bring up blood and had constant and repeated hemoptyses for the three-year period. The amount of blood varied from streaking up to what she described as over a cupful of bright red blood, which she felt came from the left side, and she had the feeling that if she exerted pressure on the left side it seemed to help control the bleeding. She also had for one year a history of very severe backache on the left side for which she had consulted an orthopedic surgeon and was given advice and treatment. She felt that the only factors which caused bleeding or aggravated it, were exertion of any kind, jolting or jarring during a rough ride, or anything of that type, and for some reason bleeding was more marked at the time of her menstrual periods. That fact had led a doctor elsewhere to give deep x-ray therapy to the ovaries—a very startling fact when we think of it in terms of the actual pathologic process present.

(Slides.) Investigation along all the usual lines did not give a definite diagnosis, but as the one slide of this chest will show, in the region of the left hilum there is an area of increased density and some calcification. Thoracotomy revealed an area of induration in the apical segment of the left lower lobe, with massive adhesions, indicating an old pleuritic process all the way round, particularly posteriorly, no doubt explaining her back pain. Because of the fact that the bronchogram had suggested some degree of cylindrical bronchiectasis in the basal segment of the lobe, I did a left lower lobectomy. I am glad to say that she made an excellent recovery.

The pathologic report on the specimen described dilatation of the bronchus to the apical segment of the left lower lobe, to form an irregular cavity, some $2\frac{1}{2}$ by $1\frac{1}{2}$ cm., with a rough lining of granulation tissue and complete destruction of the normal epithelium. cm." The summary was, "Broncholith, chronic bronchiectasis."

This cavity contained "a hard calcified dark, calculus-like mass measuring 1 by 0.5 by 0.3

In the last two years she has had no further symptoms and no trouble. I feel that this is perhaps a slightly unusual case; I think, if I interpreted Dr. Schmidt's remarks correctly, he did not have any in the apical segment of the left lower lobe, and the left side is apparently less common. It may be noted that this patient had two calcified glands which were demonstrated at operation, one of which can be seen in the left hilar region. That may well give a clue to the comments Dr. Head has made regarding etiology.

DR. ANDRE COURNAND, New York.—I think that the role of tuberculosis in broncholithiasis should not be minimized. In 1933 with Dr. Oswald Jones we published a paper on "The Shrunken Pulmonary Lobe With Chronic Bronchiectasis" in which it was demonstrated that the retraction of a middle lobe was probably related to the development of a primary tuberculous complex in the following manner: (1) primary parenchymatous lesion; (2) caseous lymph node located around the middle lobe bronchus with invasion of the latter and secondary contraction of the middle lobe. The only clinical evidence of such a process is the presence of calcifications at the hilum, which may eventually be seen to protrude into the lumen of the bronchus. In such a case, which presented a syndrome of a shrunken middle lobe and bronchiectasis without suppuration, the patient coughed up shortly after bronchoscopy two small pieces of calcium, which were no longer visible on the films. Sixteen years later this man 61 years of age was doing very well and, on the basis of that experience, I wonder whether in similar cases the lobe should always be removed.

DR. SCHMIDT.—I wish to thank the discussers. I did not think it would be necessary to try to convince anyone in this group that he should explore peripherally placed indeterminate pulmonary lesions which present the findings of obstructive pneumonia. I gathered from Dr. Head's discussion that he was somewhat reluctant about exploring this type of lesion. It has been our experience that a fairly large percentage of these lesions have proved to be due to bronchiogenic carcinoma and consequently we have found it far safer to perform exploratory thoracotomy.

In answer to Dr. Cournand's question, it is possible for caseous tuberculous lymph nodes, as well as anthracotic lymph nodes, to erode into the tracheobronchial tree. In a fairly large percentage of cases in which a broncholith has obstructed a bronchus for a long period of time, one finds that bronchiectasis has developed in that portion of the bronchial tree distal to the site of obstruction.

THE RESULTS OF SURGERY IN BRONCHIECTASIS

ADRIAN LAMBERT, M.D., F.A.C.S.
NEW YORK, N. Y.

DURING the years 1939 to 1945 inclusive, 103 operations for bronchiectasis were performed on 103 patients on the Chest Division of Bellevue Hospital. These cases have been analyzed and grouped according to the extent of the disease process and the type of associated pathology to show the significance of these factors in the surgical treatment of this disease.

Cases in which tuberculosis was demonstrated in the specimen removed have been omitted from this series. Similarly, cases in which the primary disease process was a lung abscess have not been included. Acute suppuration associated with bronchiectasis includes those cases which on microscopic examination revealed active pneumonia and acute inflammation and are differentiated from the group in which chronic inflammatory changes, interstitial fibrosis, plasma cell and lymphocytic infiltration with pneumonitis were present, without evidence of acute inflammation. Cases without acute suppuration are further divided into those with minimal parenchymal changes and those showing marked fibrosis and chronic inflammatory reaction associated with the bronchiectasis. Such pathologic classification has been made to show a possible correlation with clinical bronchiectasis. Clinically it may be impossible to differentiate bronchiectasis with acute suppuration from bronchiectasis with marked chronic inflammatory changes. Our study demonstrates the importance of recognizing the difference in the results of surgery between the two groups, as well as the bearing that the extent of the disease has on the prognosis.

Fifty-five lobectomies were performed on 54 patients for bronchiectasis uncomplicated by acute suppuration, and 30 lobectomies on 28 patients were for bronchiectasis associated with acute suppurative disease. Of the 55 patients without acute suppuration 17 had minimal parenchymal changes in the lung, and 38 had varying degrees of marked fibrosis, chronic inflammation, and interstitial pneumonitis. The remaining 21 patients had bronchiectasis involving the entire lung on one side for which pneumonectomy was performed. Of these, 8 had pathologic evidence of acute suppuration with pneumonia and abscess formation, and 13 had no acute suppurative disease but included cases varying from marked chronic inflammation and fibrosis to minimal parenchymal disease.

One patient with bronchiectasis underwent two lobectomies including the lower lobe on the left side and the lower and middle lobes on the right side. Two patients in the group in which associated acute suppurative disease was present had two lobectomies. In the first patient the upper lobe on the left side was removed followed by the lower on the same side; in the other the right lower lobe was removed in two operations.

Of the resections for uncomplicated bronchiectasis without acute suppuration 5 were for disease in the upper lobes, 31 for disease in the lower lobes, and 3 for disease in the middle lobe. In 15 patients lower and middle lobectomy

Read at the Twenty-ninth Annual Meeting of The American Association for Thoracic Surgery, New Orleans, La., March 29-31, 1949.

was performed on the right, or lower lobectomy and lingulectomy on the left, and in one patient the right upper and middle lobes were removed at one operation.

For bronchiectasis associated with suppurative pneumonitis lobectomy was performed for disease in the upper lobes in 8 cases, lower lobes in 13 cases, middle lobe in 2 cases. Right upper and middle lobes were removed in 3 instances, right lower and middle in 2 instances, and right upper and part of right lower in 2 instances.

Table I represents an analysis of the mortality in the four groups in which lobectomy was performed in the first two, and pneumonectomy in the third and fourth groups.

The average duration of symptoms in the first group, that is, bronchiectasis uncomplicated by acute suppurative disease, was 9.5 years, indicating that the disease may frequently be of long standing without manifesting symptoms sufficient to bring the patient to surgical intervention. In contrast, in the second group in which bronchiectasis was accompanied by suppurative disease, the symptoms were present for only 2.7 years. In the third and fourth groups in which the disease had progressed to involve the entire lung the symptoms were also present for a longer period in the cases in which no acute suppuration was present than in the group in which the process was more acute.

The age incidence in uncomplicated bronchiectasis was appreciably lower than in bronchiectasis with associated acute suppurative disease, regardless of whether the disease was limited to one lobe or one lung. There was a higher incidence of patients under 30 years of age in cases without acute suppurative disease and a lower incidence above 40 years of age, which included bronchiectasis in one or two lobes or involving the entire lung (Table I).

TABLE I. MORTALITY IN SURGERY FOR BRONCHIECTASIS

PATHOLOGY	NUMBER OF OPERATIONS	AGE GROUPS		AVERAGE DURATION OF SYMPTOMS (YR.)	OPERATION	MORTALITY	
		UNDER 30 YR.	OVER 40 YR.			OPERATIVE* (PER CENT)	HOSPITAL† (PER CENT)
Bronchiectasis in one or two lobes, without acute suppurative disease	55	32	7	9.5	Lobectomy	3.6	5.5
Bronchiectasis in one or two lobes, with acute suppurative disease	30	0	0	2.7	Lobectomy	6.7	10.0
Bronchiectasis without acute suppurative disease, involving an entire lung	13	7	3	7.1	Pneumonectomy	23.1	23.1
Bronchiectasis with acute suppurative disease, involving an entire lung	8	0	5	3.8	Pneumonectomy	37.5	37.5

*Died during first postoperative month.

†Died during prolonged hospital stay.

This table shows that younger individuals are susceptible to infection in the origin of their bronchiectasis. They are, however, less susceptible to acute pulmonary suppuration once the bronchiectasis has developed, and the older the patient the more likely is bronchiectasis to be associated with suppurative disease.

In stating mortality percentages, operative mortality refers to deaths occurring within the first month following operation, and the term hospital mortality refers to all cases in which death occurred during the prolonged hospital stay following operation. This distinction has been made to differentiate the mortality directly referable to operation from that which may have occurred incidental to the disease, such as brain abscess, and that may develop months after operation while the patient is still in the hospital, because, as frequently occurs in city hospitals, he has no other place to go.

It will be noted from Table I that following lobectomy the mortality is almost twice as great in the group in which bronchiectasis is complicated by acute suppuration than in the similar group without suppurative disease. The mortality in these two groups corresponds directly with the incidence of post-operative fistula, pleural infection, and wound infection—in short, with the development of complications. It is important, therefore, in this respect to bear in mind the pathology as directly related to the incidence of complications and hence indirectly to the mortality of resection as an operation for this disease.

Table II shows the causes of death in the four groups in relation to the pathology present and the type of surgery performed. In the first and second groups 33 per cent of deaths were considered due to infections induced by secretions. In the group treated by pneumonectomy half of the deaths were referable to secretions. It is interesting to note that in the first two groups

TABLE II. CAUSES OF DEATH

PATHOLOGY	OPERATION	CAUSE OF DEATH	NUMBER OF CASES
Bronchiectasis in one or two lobes, without acute suppurative disease	Lobectomy	Hemorrhage	1
		Bronchopneumonia	1
		Brain abscess two months post-operatively	1 [*]
Bronchiectasis in one or two lobes, with acute suppurative disease	Lobectomy	Hemorrhage one hour postoperatively	1
		Pneumonia in opposite lung	1
		Brain abscess three months postoperatively	1 [*]
Bronchiectasis involving an entire lung, without acute suppurative disease	Pneumonectomy	Hemorrhage on the table	1
		Anoxia on the table from profuse secretions	1
		Empyema with bronchopleural fistula	1
Bronchiectasis involving an entire lung, associated with abscess, acute suppurative disease, pneumonia	Pneumonectomy	Hemorrhage on the table	1
		Bronchopneumonia in opposite lung	1
		Urinary shut down after transfusion	1

*Hospital mortality.

there was a similarity in the causes of death, namely, hemorrhage, immediate postoperative infection, and late postoperative manifestation of infection (brain abscess) (Table II).

Table III presents an analysis of mortality from postoperative complications in relation to the operative technique. It will be seen that the incidence of bronchopleural fistula following lobectomy is lower in cases in which individual ligation was employed than in those handled by clamp or tourniquet technique. This was true in both bronchiectasis and bronchiectasis associated with suppurative disease. Of all lobectomies performed in this series the incidence of fistula was higher (63.6 per cent) in upper lobectomies than in lower lobectomies (37.8 per cent). In cases in which two lobes or one lobe and a segment of another (lingula) lobe were removed the incidence of fistula was 71.4 per cent.

The higher percentage of fistula in this series has subsequently been lowered because of greater care in handling the stump, more frequent use of individual ligation of the stump with pleuralizing of the bronchial stump and the reduction of the acute suppurative element in the lung that is to be removed before bringing the patient to operation.

TABLE III. MORTALITY FROM POSTOPERATIVE COMPLICATIONS IN RELATION TO TECHNIQUE AND TO EXISTING PATHOLOGY

PATHOLOGY	NUMBER OF CASES	OPERATION	TECHNIQUE	COMPLICATIONS			MORTALITY (PER CENT)
				FISTULA (PER CENT)	PLEURAL INFECTION (PER CENT)	WOUND INFECTION (PER CENT)	
Bronchiectasis in one or two lobes, without acute suppurative disease; with minimal parenchymal changes.	17	Lobectomy	Individual ligation	4	25.0	25.0	0
			Clamp	1	100.0	100.0	0
			Tourniquet	12	45.5	45.5	18.2
Bronchiectasis in one or two lobes, with fibrosis of parenchyma and extensive chronic inflammatory changes.	38	Lobectomy	Individual ligation	10	20.0	40.0	20.0
			Clamp	7	14.3	28.6	0
			Tourniquet	21	57.1	27.1	14.3
Bronchiectasis in one or two lobes, with acute suppurative disease.	30	Lobectomy	Individual ligation	4	25.0	50.0	25.0
			Clamp	7	50.0	100.0	0
			Tourniquet	19	82.4	94.1	53.8
Bronchiectasis involving an entire lung, with acute suppuration; abscess; pneumonia.	8	Pneumectomy	Individual ligation	4	33.3	66.6	0
			Clamp	2	100.0	100.0	0
			Tourniquet	2	0	100.0	0
Bronchiectasis involving an entire lung with chronic infection and marked or minimal parenchymal fibrosis.	13	Pneumectomy	Individual ligation	6	40.0	60.0	20.0
			Clamp	0	—	—	—
			Tourniquet	7	33.3	33.3	20.0

Pleural infection following lobectomy in the uncomplicated bronchiectasis group was highest when treated by tourniquet and in bronchiectasis associated with suppurative disease was lowest when treated by individual ligation. Wound infection was highest in all patients with suppuration operated on by both lobectomy and pneumonectomy, in which the tourniquet technique was used. In the pneumonectomy group the incidence of fistula and pleural infection ran parallel, as might be expected. This group represents extremely serious cases in which the amount of sputum at operation averaged about 100 c.c., a reduction from an average of 215 c.c. on admission. Some of these patients were so desperately ill that surgery was used as a last resort in an attempt to interrupt a cycle in which the patient's condition not only became progressively worse over a period averaging six years, but he came to surgery in a condition of malnutrition, wasting, and chronic sepsis from which he could not recover without surgical intervention. These patients, therefore, constitute largely a salvage group and differ from those cases in which the disease had not reached the extent of that in the pneumonectomy group (Table III).

These three groups were also studied in relation to the incidence of sputum as presented in Table IV. It will be noted that in the bronchiectasis group with acute suppuration treated by lobectomy and in the bronchiectasis group without acute suppuration treated by lobectomy, the amount of sputum on admission was approximately the same, but in the second group it was possible before operation to reduce the amount of sputum to almost one-half of that in the first group. Also, the amount of sputum in the cases in which bronchiectasis involved an entire lung was more on admission and at operation than in those in which similar disease was localized to part of the lung. Average sputum on admission of patients who died following pneumonectomy was 400 c.c. daily, which contrasted markedly with 130 c.c. daily admission sputum in the group that survived. In these cases sputum was reduced to 130 c.c. and 85 c.c., respectively, at the time of operation. The absence of acute suppuration, therefore, may best be determined by the relative sputum reduction following preoperative treatment rather than the amount that is present on admission (Table IV).

The practical application of this observation includes delaying resection until the sputum is brought to the minimum in order to reduce the acute suppurative element as much as possible. Preoperative measures included postural drainage with bronchoscopic aspiration in resistant cases. Chemotherapy was employed to reduce secretions and was determined by the type of bacterial flora present in cultures. Sulfadiazine was used at first; later it was replaced by penicillin, as being more specific for more organisms and less toxic for the patient. Recently streptomycin has been added to the specific drugs that may be employed. It is reserved for patients having predominantly gram-negative organisms that are streptomycin sensitive. Patients in whom the organisms present are penicillin resistant are now treated with sulfadiazine. Patients who become allergic to sulfadiazine may be treated with sulfathiazole to avoid the specific sensitivity. Cases of overwhelming anaerobic infection frequently will

TABLE IV. INCIDENCE OF SPUTUM IN RELATION TO MORTALITY

PATHOLOGY	NUMBER OF OPERATIONS	OPERATION	AVERAGE AMOUNT OF SPUTUM ON ADMISSION (C.C.)	TYPE OF SPUTUM BEFORE OPERATION				AVERAGE AMOUNT OF SPUTUM AT OPERATION (C.C.)	HOSPITAL MORTALITY			
				FOUL		NONFOUL			FOUL		NONFOUL	
				NUM- BER	PER CENT	NUM- BER	PER CENT		NUM- BER	PER CENT	NUM- BER	PER CENT
Bronchiectasis without acute suppurative disease in one or two lobes	55	Lobectomy	120	20	36.4	35	63.6	40	1	5.0	2	5.7
Bronchiectasis with acute suppurative disease in one or two lobes	30	Lobectomy	125	10	50.3	11	40.7	70	2	12.5	1	9.1
Bronchiectasis without acute suppurative disease involving an entire lung	13	Pneumonec- tomy	210	7	53.8	6	46.2	80	3	42.9	0	0
Bronchiectasis with acute suppurative disease, involving an entire lung	8	Pneumonec- tomy	220	7	87.5	1	12.5	135	3	42.9	0	0

respond to oxygen therapy administered with high concentration mask so as to reduce the sputum and convert it from foul to nonfoul variety, that is, from aerobic to anaerobic flora.

Aerosol administration of penicillin or streptomycin has been found of value in reducing secretions when intramuscular injection did not bring about a high enough concentration to affect the organisms. This may be used in conjunction with intramuscular therapy.

The incidence of foul or occasionally foul sputum was found to be lowest in the uncomplicated bronchiectasis group, becoming progressively greater depending upon the presence of suppuration and the degree of lung involvement. In analyzing the mortality in these cases it was found that there was almost no difference between the percentage of deaths in cases with foul sputum and those with nonfoul sputum in simple uncomplicated bronchiectasis. With the presence of suppuration, however, the mortality became appreciably greater in cases with foul sputum, so that when the entire lung was involved the mortality was limited to those patients having foul sputum.

In a total of 106 operations on 103 patients for bronchiectasis there were 12 deaths from various causes as explained in Tables I through IV. Seventy-eight of the 91 survivals were observed over an average follow-up period of four years, and grouped according to original pathology.

Of the 78 cases followed, 65 were lobectomies and 13 were pneumonectomies. Of the 65 lobectomies, 16 patients had other lobe involvement with bronchiectasis. Twelve of these were not resected at a later date, and 2 patients died; 4 had further surgery for other lobe bronchiectasis. Of the 12 cases, 11 fell into the group with chronic inflammatory changes and fibrosis without acute suppuration before operation, none was in the group in which minimal changes were found associated with the bronchiectasis, and one was in the group with acute suppuration.

Nine patients had symptoms from residual bronchial stump, 1 of which was following pneumonectomy and the others following lobectomy. The 1 pneumonectomy patient and 5 of the lobectomy patients underwent surgical revision of the stump. Three patients still have stumps from which they continue to have symptoms. Two of these refused operation and in 1 case resection was unsuccessfully attempted. Six of the 8 stumps following lobectomy were in the group in which bronchiectasis was associated before operation with acute suppuration, and the other 2 were in the group that showed chronic inflammatory changes. There was no stump in the group in which minimal inflammatory disease was associated with the original bronchiectasis. The 1 pneumonectomy coming to stump resection had previous chronic inflammatory changes associated with the bronchiectasis. In keeping with these observations, 7 of the 9 residual stumps were resected with tourniquet technique and 2 were divided between clamps. None was handled by individual ligation. This emphasizes the importance of bringing these cases to the operating table with as little suppurative disease as possible so that the surgeon will be able to utilize this type of technique. To wait until acute suppurative changes are present which develop

into the chronic inflammatory type with associated fibrosis will result in higher mortality and higher incidence of complications. Among these are listed the revision of a long bronchial stump as seen in 9 cases of this series.

Sinusitis has been present in 14 cases that were followed postoperatively, and all are lobectomy patients. No patients treated by pneumonectomy were bothered by sinus trouble after operation. Of the 14 patients developing sinus disease, 1 developed positive sputum; 9 had less than $\frac{1}{2}$ ounce of sputum a day; 4 had between 2 and 4 ounces of sputum a day. Of these 9 patients, 1 had an episode of pneumonia five months postoperatively, 1 had lingulectomy postoperatively with continued sputum of 15 c.c. due to sinus disease, and 2 had occasional flare-ups of sinusitis at which time their sputum increased to 100 c.c. and 40 c.c., respectively. In both these cases the tourniquet technique was used. Of the 4 patients having between 2 and 4 ounces of sputum daily, 2 had further bronchiectasis in the middle lobe, and 2 had residual stumps following lobectomy with tourniquet technique for which they refused further resection.

Of the 65 lobectomies followed, 43, or 66.2 per cent of lobectomies, had sputum of $\frac{1}{2}$ ounce or less postoperatively; 10, or 15.4 per cent, had continued sputum daily of 1 ounce and more. Of these 10 cases, 4 had persisting sinus disease, 6 had further bronchiectasis without operation, of which one was followed to her death from bronchiectasis, and 3 had stumps from the initial procedure that continued to give them symptoms. The sputum in the above groups is exclusive of those patients who developed tuberculosis.

Of the 13 cases treated by pneumonectomy none suffered from sinus disease, none had significant sputum postoperatively, and only 1 had minimal sputum (1 teaspoonful).

Symptoms of pain were present following pneumonectomy in 2 cases. In 1 case metastatic carcinoma of the ribs developed; this patient may have had carcinoma of the lung although it was not noted on section at the time the specimen was removed and the primary site was never determined. The second case was complicated by empyema and has not been seen since thoracoplasty. Two lobectomy patients had pain that persisted on follow-up, both following left lower lobectomy. One of these had a residual stump for which she refused further operation.

Four lobectomy patients had dyspnea on exertion with some shift of the mediastinum following operation; in 5 pneumonectomies moderate dyspnea was present postoperatively, and 2 developed scoliosis. Ten pneumonectomies required thoracoplasty procedures, a total of 40 operations being performed on the 13 patients in the pneumonectomy group following resection. Nine lobectomy patients required thoracoplasty, of which 4 were for tuberculosis. Of the remaining 5, 4 had upper lobe lobectomies and the fifth had previously had an upper lobe lobectomy and needed thoracoplasty for the empyema that followed lower lobectomy on the same side.

Ten patients, or 12.8 per cent of the 78 survivals followed for an average of four years, developed tuberculosis postoperatively. Of this number, 2 died of tuberculosis in seven months and sixteen months, respectively. Of the remaining 8 in this group, 5 still have positive sputum; 3 have negative sputum.

In 6 of the 10 cases the tuberculosis developed within a year; in 4 cases the disease was noted after sixteen, twenty-three, twenty-four and thirty months, respectively. In 1 case the disease developed in the pleural space and the patient had never had positive sputum; in all the other cases tubercle bacilli were present in the sputum.

The development of tuberculosis in these 10 cases had no relation to the site of the bronchiectasis. In 1 case the right upper lobe had been removed, in another case, the right upper and middle lobes; in 4 cases the lower and middle or lingula, and in 2 the lower lobe was removed; in 2 cases pneumonectomy had been performed. In 6 cases the tourniquet technique was employed, in 2 cases, individual ligation, and in the 2 remaining cases, division between clamps was the technique used.

The possible mechanism of infection in these cases is worthy of consideration. Undetected tuberculosis may have been present in the lobe removed and spread to the remaining lung tissue at operation. Second, the disease may have been present at the hilum and tuberculous tissue may have been cut across at operation, thus lighting up the previously quiescent disease. Third, the disease may have been lighted up in the remaining lung as a result of overdistention following resection. Last, airborne or contact infection must be considered on a service where it is impossible, owing to overcrowded conditions, to segregate tuberculous patients before the diagnosis has been confirmed.

Of these possibilities, the first is the least likely. If sufficient ulceration were present in the lung removed to cause spread, it should be noted on routine pathologic examination. That tuberculosis may have been present at the hilum is suggested by the high incidence of the development of the disease following tourniquet resection.

In this series of cases, individual ligation was less universally employed than is now the case. Furthermore, the extent of the disease at the hilum required the employment of the tourniquet technique. Of the 2 individual ligations, 1, a pneumonectomy, was the case in which the disease developed in the pleural space following empyema and bronchopleural fistula; in the other, the left lower and lingual lobes were removed together, suggesting division of lung tissue. Dividing the hilum between clamps implies the sectioning of pulmonary tissue and the possibility of cutting across tuberculous tissue. In 1 case sputum became positive after attempted removal of the stump following tourniquet resection, and in another the sputum became first positive, then negative, then positive again after further stump excision eighteen months after the original operation.

Overdistention as a factor in the development of tuberculosis was considered probable in 5 of the 10 cases. In 4 of these the disease developed in the remaining portion of the resected lung. In retrospect, after reviewing the preoperative roentgenograms, it is felt that 2 of these patients may have had unrecognized chronic pulmonary tuberculosis, although only one had a history of previous

tuberculosis. In one case tuberculosis developed on the opposite side after pneumonectomy. In this case enlarged glands were present at the hilum at the time of operation but there was no suggestion made that they were tuberculous; however, sections were not taken at operation.

Of the remaining 5 cases in which tuberculosis developed, 3 had residual stumps following operation. One cleared up with reduction of sputum, a second developed positive sputum after attempted resection of the stump, and the third developed positive sputum after resection, became negative, and then positive again after excision of the stump. In the first of these, resection was by division between clamps, and in the other 2 the tourniquet technique was employed. The fourth was the pneumonectomy case in which tuberculosis developed in the pleural space following bronchopleural fistula, necessitating multiple operations to obliterate the sinus and the pleural cavity. In the fifth case a tourniquet stump repair was done for resection of the left lower and lingula of the left upper lobe which was followed by the development of large amounts of sputum due to tuberculosis of the right side, developing first in the right upper lobe and then spreading to the other side, and to the intestinal tract, resulting in death five months after the disease was diagnosed by the presence of tubercle bacilli in the sputum.

From the analysis of these 10 cases it is evident that several factors could account for the development of tuberculosis following operation for bronchiectasis. All cases should be carefully evaluated by x-ray examination for the possibility of unrecognized tuberculosis before coming to operation. If there is a suggestion of inactive tuberculosis in the remaining lobe following lobectomy or of the opposite lung following pneumonectomy, early thoracoplasty is advisable to prevent overdistention and the possible reactivation of remaining tuberculosis.

These 106 operations for bronchiectasis represent lobectomies and pneumonectomies done during the years 1939 to 1945. Segmental resection in this study was limited to cases in which the lingula of the upper lobe was removed in conjunction with the upper or lower lobe on the left side. In 1 case part of the upper and lower lobes on the right side were removed but the modern type of segmental resection was not employed.

Since then segmental resection has been employed more frequently for bronchiectasis. The principle of the operation is to remove that portion of the lobe in which bronchiectasis is present and preserve as much of the functioning lung tissue as possible. This implies the removal of only the diseased tissue, so that there will not be a lack of functioning lung tissue as the result of pulmonary resection, nor unnecessary removal of normal lung for lack of any easy anatomic cleavage.

The importance of the present study of cases lies in the bearing of the pathology in the resected lung on the postoperative course. The fundamental principles that become obvious in these cases apply to whatever operative technique is employed. Control of the acute suppurative and chronic inflammatory factors in bronchiectasis is as important for a segmental resection as for a lobectomy or pneumonectomy. Suppuration as encountered in this series represents bron-

chiectasis in its most serious form. The advanced stages of suppuration found in some of these patients implied an ignorance of the eventual outcome of the disease by the patient and the doctor.

The mortality in this group, which is so much higher than in the group that presented no acute suppuration, does not represent the same mortality as for uncomplicated bronchiectasis. The surgical risk will be determined by the extent and type of associated suppurative disease.

With the emphasis being laid on segmental resection at the present time it is well to bear in mind that this operation is done with the individual type of technique. It is therefore important to eliminate the suppurative factor as much as possible before bringing the patient to operation. As more experience in this type of technique has been obtained both the mortality and the incidence of complications have been decreased.

In summary, it may be said that resection for bronchiectasis is a less formidable procedure in those cases in which the disease is not associated with acute suppuration as determined by the incidence of complications and mortality following resection. That the incidence of fistula is lower in lobectomy for uncomplicated bronchiectasis than in bronchiectasis associated with suppurative disease focuses the attention on the hilum where fistula occurs postoperatively. When suppurative disease is present, the hilum is involved in the inflammatory process to a greater degree than in simple bronchiectasis where there is no inflammation in the periphery and therefore no involvement of the hilum. Chemotherapy has been of great assistance in reducing inflammation at the hilum, and therefore should be responsible for further lowering the morbidity and mortality of the operation.

As is true of any serious disease, surgery is employed for the purpose of relieving the patient of his symptoms. In bronchiectasis this is ideally performed in the uncomplicated stage of the disease. If the patient has passed the point of ideal operability, we still feel that he ought to be given the benefit of surgery, realizing at the same time that although the risk is greater, the results will be correspondingly more gratifying.

REFERENCES

- Brunn, H.: The Technique of Lobectomy in One Stage, *Surg., Gynec. & Obst.* 55: 616, 1932.
 Janes, R. M.: Surgical Treatment of Bronchiectasis, *Brit. J. Surg.* 21: 257-276, 1933.
 Robinson, W. L.: Bronchiectasis: Diagnosis and Prognosis in Relation to Treatment, *Brit. J. Surg.* 21: 302-312, 1933.
 Bendove, R. A., and Gershwin, B. S.: Morphologic Varieties of Bronchiectasis in the Adult: Their Probable Pathogenesis and Clinical Differentiation, *Arch. Int. Med.* 54: 131-144, 1934.
 Roberts, J. E. H.: Discussion on Lobectomy in Pulmonary Tuberculosis, *J. THORACIC SURG.* 5: 141, 1935.
 Churchill, E. D.: Bronchiectasis: Physical and Psychological Manifestations, *New England J. Med.* 218: 97-101, 1938.
 Chapman, J., and Hoskins, H.: Morphological Varieties of Bronchiectasis in Relation to History, Symptoms and Signs, *Am. Rev. Tuberc.* 43: 512-520, 1941.
 Ogilvie, A. G.: Natural History of Bronchiectasis; Clinical, Roentgenological and Pathological Study, *Arch. Int. Med.* 68: 395-465, 1941.
 Blades, B., and Graham, E. A.: Surgical Treatment of Bilateral Bronchiectasis, *Surg., Gynec. & Obst.* 75: 457-464, 1942.
 Meade, R. H., Kay, B., and Hughes, F. A.: A Report of 196 Lobectomies Performed at Kennedy General Hospital Chest Surgical Center From 1943 to 1946 With One Death, *J. THORACIC SURG.* 16: 16-26, 1947.

(See discussion on page 265.)

THE SURGICAL TREATMENT OF BILATERAL BRONCHIECTASIS

FREDERICK G. KERGIN, M.S., F.R.C.S.

TORONTO, CANADA

ALTHOUGH the first report of bilateral lobectomy for bilateral bronchiectasis was that of Eloesser in 1933, no report of any considerable series of cases has appeared in the intervening years. In spite of the number of patients available, for one-third of bronchiectatic patients have bilateral disease, and the morbidity and mortality to which they are subject, thoracic surgeons appear to have been cautious in undertaking bilateral resection.

In 1942 Blades and Graham reported 4 bilateral resections with 1 death. In the same paper they mentioned 12 other cases of bilateral bronchiectasis, subjected to unilateral resection, with 3 deaths. As recently as 1947, Biscard and Swenson reported 4 successful cases of bilateral resection, and from the literature to that date, could collect only 37 further cases, with 5 deaths. Strieder, Overholt, Laird, and others in discussing bronchiectasis have referred to small groups of patients in whom bilateral resections have been done, and there have been a number of individual case reports.

Because conclusions in regard to mortality rate and expectation of cure formed on the basis of reports of small groups of cases from various centers are likely to be misleading, it has seemed worth while to study a group of patients with bilateral bronchiectasis, who have been treated at the Toronto General Hospital and Hospital for Sick Children, Toronto.*

The group consists of 58 patients of whom 27 have had unilateral operations and 31 have had bilateral operations. A total of 94 excision operations have been done on these patients, with 4 deaths, an operation mortality rate of 4 per cent, and a patient mortality rate of 7 per cent. The 4 deaths all followed the second side of a bilateral resection; therefore, in the 58 patients there has been no mortality following operation on the first side, but in the 31 patients who have had excision on the second side there have been a mortality of 13 per cent. It is of interest that these 4 deaths occurred in the first 13 cases. By attention to certain technical details, which will be discussed below, it has been possible to complete the last 18 consecutive bilateral resections without a death.

Forty-eight of the patients in the series were treated at the Toronto General Hospital. In age they varied from 14 to 43 years, with an average of 22 years. The other 10 were treated at the Hospital for Sick Children and varied in age from 4 to 14 years, with an average of 10 years. The 4 deaths all occurred in the older group. The oldest patient on whom bilateral resections have been completed was 28 at the time of the second operation. The younger patients,

From the Department of Surgery, University of Toronto, and the Toronto General Hospital.

Read at the Twenty-ninth Annual Meeting of The American Association for Thoracic Surgery, New Orleans, La., March 29-31, 1949.

*The patients in the Toronto General Hospital group were operated upon by Dr. N. S. Shenstone, Dr. R. M. Jones, and the author, and in the Hospital for Sick Children group by Dr. R. M. Jones, Dr. F. R. Wilkinson, and the author. The author is grateful to these surgeons for permission to include their cases in the series.

and in particular the children, stand extensive resection better than do the older group, but no definite conclusion has been reached in regard to the age limit for bilateral resection. One patient, 39 years of age, with extremely severe disease involving the right middle and lower lobes and the left lower lobe and lingula has had a resection on the right and will shortly be subjected to operation on the left.

Patients with bilateral bronchiectasis may be divided into two groups on the basis of the degree of involvement of the second side. The first group have severe or moderately severe disease of the first side, with slight or minimal disease of the second side. In the second group the disease on both sides is severe or moderately severe. The policy which has been followed has been to operate only on the more severely diseased side in Group 1, but to do bilateral resections in Group 2.

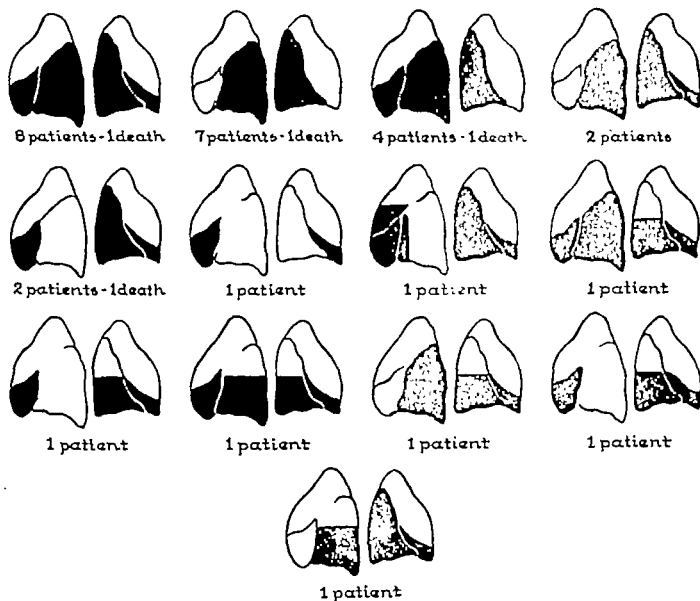
In this series, 15 patients showed minimal disease on the second side and were treated by unilateral resections. Eleven now have no symptoms, and consider themselves cured; 3 are greatly improved, complain only of slight cough, and have less than 1 ounce of sputum per day. These 14 patients have a satisfactory result from their treatment, but the fifteenth patient demonstrates that the degree as well as the extent of the disease on the second side is important. She has had a resection of the basic divisions and the lingual segment on the left, and a bronchogram shows that there is no residual disease on that side. On the right the disease is confined to the anterior basic division, but in that bronchus is rather severe. The patient still has 3 ounces of purulent sputum per day; she will require a resection of the involved segment.

The second group of patients, 43 in number, have had severe or moderately severe disease in both lungs. Very gross disease has been considered an indication for operation, rather than a contraindication, provided that the upper lobes with the exception of the lingual segment were healthy. These patients have all been improved by the first operation; sputum has usually been reduced by one-third or one-half, the patients have gained weight and had considerable improvement in their sense of well-being. At times, it is tempting to both patient and surgeon to accept a degree of improvement and avoid the risk of a second operation, but as these patients are followed, some show relapse, with increase in their symptoms, and all continue to be subject to the disability and dangers of residual bronchiectasis. For that reason, before any surgical treatment is undertaken, these patients agree to accept the hazards of bilateral resection.

In 31 of these 43 patients, bilateral resections have been completed and 4 others are awaiting operation on the second side. The other 8 patients have had unilateral resection. One patient moved to a distant part of the country after the first operation and was lost to the follow-up, 2 patients are so much improved after the first operation that they are satisfied with their result and have carried on for seven and five years, respectively, 3 patients have refused the second operation in spite of fairly severe persisting symptoms, and 2 of the earliest cases, on more complete bronchographic examination of the second side showed disease too extensive for resection. They should not have been operated

upon, and under the present rigid rule that no operation will be done until all segments of both lungs have been clearly demonstrated by bronchography, they would not have been accepted for surgical treatment.

It is the study of the 31 patients who have had bilateral resections which illustrates the problems in the complete surgical treatment of this disease. Fig. 1 illustrates the resections which have been done in these patients and indicates the types of resection, which resulted in death. The first fatality, in 1940, was seventy days after the second operation from a combination of persisting atelectasis and empyema on the side of operation. With the modern treatment of



Thirty-one patients with bilateral bronchiectasis
treated by bilateral resection.

Fig. 1.—A diagrammatic representation of the 31 bilateral resections. The black areas indicate the lobes or segments which were resected.

atelectasis by bronchoscopic suction and the use of penicillin, this death would not have occurred. The second death, in 1942, was twenty-two days after the second resection, from a brain abscess, confirmed at autopsy. Penicillin might have prevented this. The other two deaths, in 1946, emphasize the fundamental problem in this type of surgery. One was twenty-eight hours and the other fifteen hours after the second resection and both were from cerebral anoxia which had been allowed to persist during the operation.

With dissection technique, and the use of antibiotics, sepsis, although still a problem is no longer lethal. Danger to life is due mainly to one factor—cerebral anoxia. During any thoracotomy, respiratory function depends almost entirely on the contralateral lung. If that lung is diseased, and more particularly if it has been reduced to a single lobe by a previous resection, the margin of safety is obviously very narrow. Experience has shown that with proper anesthetic technique a contralateral lung consisting of a single lobe can carry a patient through an arduous resection on the second side provided the airway to that lobe remains clear. If, however, there is any obstruction to the contralateral bronchus, the patient is immediately in grave danger from cerebral anoxia. It is to be noted that this danger extends into the postoperative period to the time when the remaining lung of the side of recent operation has been expanded and taken on adequate function.

In the prevention of anoxia there are two general considerations—the first to develop maximum respiratory function before operation and the second to protect that function by preventing bronchial obstruction. The first consideration affects the choice of side for the first operation, the timing of the operations, the use of breathing exercises to develop function, and in particular to recover it after the first operation; the second consideration implies certain measures before, during, and after operation to reduce the amount of sputum and to ensure that it is evacuated rapidly from the bronchial tree.

Because, as pointed out previously, the danger of anoxia is greater during the second resection because of the reduction of the contralateral lung by the previous lobectomy, in those cases in which there is any appreciable difference in the extent of the disease on the two sides, it has seemed reasonable to do the lesser resection first, so that, at the critical second operation, there will be a larger functioning lung. This policy has been followed in the latter half of the series.

Before undertaking the second resection, time must elapse to allow maximum functional recovery of the first side, and also to take full advantage of the improvement in general condition of the patient. Six months appears to be the minimum interval; more commonly in this group the interval has been nine months to one year. The importance of breathing exercises in recovering and increasing function cannot be overemphasized.

Sinus disease is cleared up as far as possible before undertaking any pulmonary surgery. Before each operation every method is used to reduce and evacuate bronchial secretions. The patients sleep with the foot of the bed elevated. Supervised, effective postural drainage is carried out three times a day. Expectorants are used if the secretions are difficult to raise. Penicillin is usually given by intramuscular injection for two days, immediately before operation. Inhalation of nebulized penicillin or streptomycin, or both, has not seemed to have any special advantage. Finally, thorough postural drainage is carried out immediately before giving the preoperative sedative.

Bronchoscopic suction is occasionally done immediately before and is invariably done at the termination of the operation. All operations are performed under intratracheal ether-oxygen anesthesia with endobronchial suction as in-

dicated. For the past two years patients with bilateral bronchiectasis have been operated upon in the prone position, and this decreases, but does not completely obviate the danger of a spill of mucus into the contralateral bronchus.

Since the two fatal cases of cerebral anoxia in 1946, continuous observations of the arterial oxygen saturation have been made during the operation, in all cases, by means of an oximeter. It has been demonstrated that, provided the contralateral bronchi can be kept clear by suction, the arterial oxygen saturation can be restored and maintained by the frequent application of positive pressure. The greatest value of the instrument is that it indicates when these measures are not effective, and gives warning of the existence of a persisting degree of anoxia which is dangerous. On two occasions the operation has been abandoned after freeing the lobe, because of inability to correct anoxia, and in each case the operation was completed satisfactorily at a later date. It is believed that, without the warning given by the oximeter, these 2 cases might have resulted in fatal cerebral anoxia.

Fig. 2 illustrates the record obtained in a patient with very gross bilateral disease during the resection of the first side. In spite of the prone position, as the lung was freed there was a very heavy spill of secretions into the trachea with resulting acute anoxia. The anesthetist was successful in clearing the obstruction by suction and following that the oxygen level shows the usual intermittent depressions and peaks due to suction and positive pressure.

Fig. 3 illustrates one of the cases in which the operation was abandoned because of inability to clear the opposite bronchi by endobronchial suction.

In the postoperative period every effort is made to keep the bronchi clear of mucus and to obtain rapid expansion of the remaining lung on the operative side. The foot of the bed is kept elevated, the patient turned every hour during the day, every second hour during the night, and persuaded to cough after the judicious use of sedatives. Expectorants are used. Atelectasis of either side is watched for constantly, and if it occurs is treated by immediate bronchoscopic suction. Closed drainage is used in all cases for two to four days, supplemented by pleural aspirations as indicated.

In this period the margin of safety in the respiratory function is exceedingly narrow. Success depends on constant supervision by well-trained nursing and resident staff who can recognize trouble and act quickly. Unless this supervision is available these patients should not be operated upon.

It is by the application of all the measures described above that it has been possible to complete the last 18 bilateral resections without a death.

In this group of patients the result of treatment must be assessed in relation to relief of symptoms and in terms of respiratory function following treatment. In regard to the former, an assessment should not be made until one year has elapsed after the second operation, because in some patients there is residual cough which slowly improves, while in others, after initial improvement there may be some degree of relapse. Of the 31 patients, 27 have survived. One, when last seen, a few months after the second operation, was well, but since then has been lost to the follow-up. Eight patients have been operated upon too recently for final assessment. Of the remaining 18, 15, or 83 per cent, have no

this increased metabolic rate she showed no unusual shortness of breath. She was prepared with propylthiouracil, operated on in the usual way, and made a good recovery. These clinical observations appear to support the broncho-spirometric observations of Kay, Meade, and Hughes which demonstrated that, provided chest wall and diaphragmatic function is good, the oxygen absorption following lobectomy may be essentially normal. The place of breathing exercises in regaining this good function is again stressed.

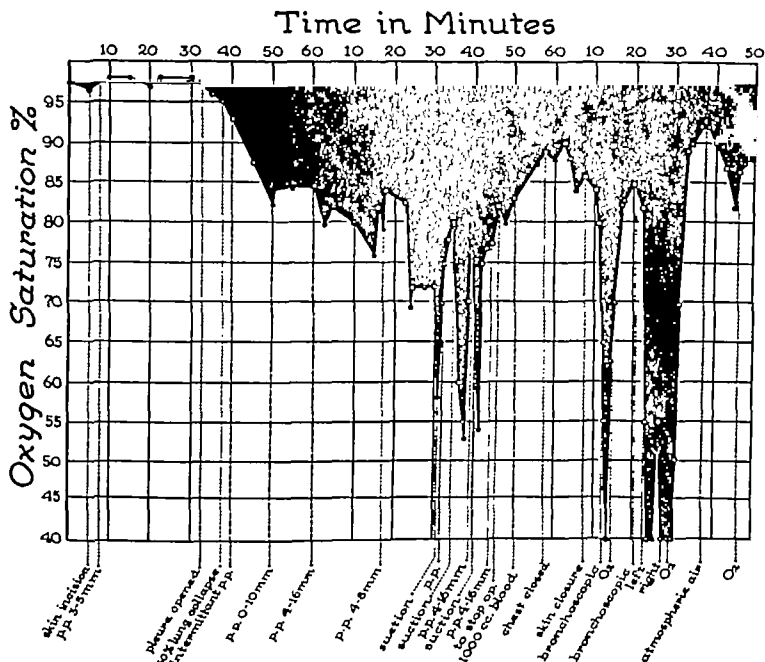


Fig. 3.—Oximeter record of first operation on R. C., 16 years of age, severe bilateral disease, resection of right lower lobe in lateral position. Repeated suction followed by positive pressure was unsuccessful in correcting anoxia. The operation was abandoned and the chest closed. The resection was successfully completed twelve days later.

Although function after these extensive resections is remarkably good, it is natural to attempt to save all healthy lung. On 13 occasions a partial lower lobectomy was done; in 11 of these all the basic segments were resected with preservation of an apparently healthy superior segment. Eight of these resections involved the left lower lobe and were combined with a resection of the lingual segments; the other 3 involved the right lower lobe and 2 of them were combined with a resection of the middle lobe. These 11 operations were performed on 10 patients, 1 girl having had bilateral segmental resection of the basic divisions.

After the 11 operations there were only 5 good recoveries. In the other 6 there were 2 empyemas, 1 persisting fistula without empyema, 2 clotted hemothoraces, and 1 case in which there was incomplete expansion of the superior segment, in spite of repeated bronchoscopic suction and pleural aspiration. Four of these 6 patients have developed bronchiectasis in the retained dorsal segments; 1 has already been resected and the other 3 will probably require resection. These 4 patients are included in the group of patients who have been treated too recently for final assessment.

Because of the high incidence of pleural complications and the tendency to develop bronchiectasis, segmental resection of all the basic divisions with preservation of the superior segment alone has been abandoned, although resection of individual basic segments is still practiced.

SUMMARY

Fifty-eight patients with bilateral bronchiectasis have been treated surgically, 27 by unilateral resection and 31 by bilateral resections. Ninety-four excision operations have been done on the group with 4 deaths, all of which occurred after an operation on the second side.

Fifteen of the patients showed marked disproportion in the distribution of the disease, in that on the second side the disease was slight. These have been treated by unilateral resection, with satisfactory result in all but 1 patient.

The remaining 43 patients showed disease which was severe or moderately severe on both sides. Within certain limitations staged bilateral resection is recommended for this group and has been carried out in 31 patients.

The hazards associated with surgical treatment have been discussed. It has been pointed out that of these cerebral anoxia is the most important, and that it is more likely to occur during or immediately after operation on the second side. Methods which have been used to prevent anoxia have been described, including the use of an oximeter during operation to provide an accurate method of following fluctuations in the arterial oxygen level.

In 18 of the 27 patients who have survived bilateral resection, the final result can be assessed. Fifteen are symptom free and all of them show remarkably little evidence of decreased respiratory function.

The practice of segmental resection of the basic divisions of the lower lobes, with preservation of the superior segment, has been abandoned because of the high incidence of pleural complications and a tendency to the development of bronchiectasis in the previously healthy superior segment.

REFERENCES

1. Bisgard, J. D., and Swenson, S. A., Jr.: Bilateral Lobectomy for Bilateral Bronchiectasis, *Arch. Surg.* 54: 483-490, 1947.
2. Blades, B., and Graham, E. A.: The Surgical Treatment of Bilateral Bronchiectasis, *Surg., Gynec. & Obst.* 75: 457-467, 1942.
3. Eloesser, L.: Bilateral Lobectomy, *Surg., Gynec. & Obst.* 57: 247-249, 1933.
4. Kay, E. B., Meade, R. H., Jr., and Hughes, F. A., Jr.: Surgical Treatment of Bronchiectasis, *Ann. Int. Med.* 26: 1-12, 1947.

5. Kergin, F. G., Bean, D. M., and Paul, W.: Anoxia During Intrathoracic Operations, *J. THORACIC SURG.* 17: 709-711, 1948.
6. Laird, R. C.: Bronchiectasis in Service Personnel, *J. THORACIC SURG.* 17: 57-61, 1948.
7. Overholt, R. H., Woods, F. M., and Betts, R. H.: An Improved Method of Resection of Pulmonary Segments, *J. THORACIC SURG.* 17: 464-477, 1948.
8. Strieder, J. W.: Surgical Aspects of Bronchiectasis, *New England J. Med.* 238: 100-112, 1948.

DISCUSSION ON "THE RESULTS OF SURGERY IN BRONCHIECTASIS" BY DR. ADRIAN LAMBERT,* AND "THE SURGICAL TREATMENT OF BILATERAL BRONCHIECTASIS" BY DR. FREDERICK G. KERGIN

DR. WILLIAM E. ADAMS, Chicago.—My remarks have to do chiefly with the management of bilateral bronchiectasis. I think Dr. Kergin has given us an excellent presentation of the problem involved. I heartily agree with the principles that he has emphasized that must be observed in management of these patients. A number of years ago Dr. Graham, in reporting on an extensive bilateral resection for bronchiectasis, brought up the question with how little lung function is life compatible. I think in his case he had resected both lower lobes, the middle and the lingula of the left upper. Since that time considerable has been learned regarding the management of suppurative diseases of the lung.

I would like to review briefly our experience with these bilateral cases. (Slides.) This first case was a woman, 28 years of age, who had had bilateral bronchiectasis for a number of years. This was before segmental resection was practiced, and in her case we resected both lower lobes, the right middle, and the lingula of the left upper. The next slide shows a microscopic section through one of the lobes involved and I might point out that making these large microscopic sections routinely has brought out evidence of tuberculosis in a number of cases which would otherwise have been overlooked in the smaller sections.

The second case was a man in his early 30's who, as you can see, has bronchiectasis of the lower lobes, the right middle and the lingula, all of which are completely collapsed; in addition, the lower part of both upper lobes is remarkably emphysematous. This patient represents the most extensive resection we have carried out; it included both lower lobes, the lingula, the right middle and a portion of the upper lobes. This patient had a vital capacity of around 3,700 c.c. in spite of the fact that he had only the upper two lobes functioning. Following operation roentgenographic appearance of the lung was about the same as before with the exception that the diaphragms were somewhat elevated; less dyspnea was experienced than before operation.

Here is the microscopic section of one of the lobes, which illustrates the type of disease present.

In reviewing our experience with these cases, there were 80 in which operation was done for bronchiectasis, 32 of the 66 being bilateral. Of the 32, in 14 the resection was completed and, unlike Dr. Kergin's experience, our main difficulty was following the first stage. The two operative deaths occurred in 2 of these bilateral cases following the first operation. There were no deaths following the second operation. Of these 14, 10 are on full activity, a number are earning their living working a full day. Of the 14 there were 8 women, and 5 have gone through normal pregnancies. This is evidence that these patients are not necessarily pulmonary cripples following bilateral resection. In view of the fact that at the present time we can use segmental resection in selected cases, we are able to do them a great deal of good.

DR. JOSEPH GORDON, Ray Brook, N. Y.—My discussion is rather a departure from the things that have already been said; I should like to pass on to you some information which is perhaps generally known but which has been helpful to me because of its presentation in familiar graphic form. We all know that the pulmonary arterial tree parallels the bronchial tree very closely, as has been shown many times. So much is this so, that even

*See page 246 for article by Lambert.

in disease processes which involve the lung it usually involves the bronchial tree and the pulmonary arterial system as well. The notable exception of this, however, is in bronchiectasis, and these illustrations I am about to show have helped me to understand the pathologic findings and the disturbance of the pathologic physiology in cases with bronchiectasis. (Slides.)

In this slide you see the familiar pattern of a normal bronchogram; the illustration on the right is an arteriogram showing a normal pattern except in the basal portion where there is evidence of emphysema. The pattern of the pulmonary arteriogram is similar to that seen in the bronchogram. During the past seven or eight years we have undertaken at Ray Brook the study of both surgical and post-mortem material by this method of contrast roentgenography. Keeping these two normal patterns in mind the next slide shows an arteriogram with most of the smaller vessels gone. On the right there is marked obliteration of the arterial tree in the presence of extensive disease and is even more so with thoracoplasty on the left. In the lower left corner you see the small divisions of the arterial tree showing a relatively normal pattern, contrasted with marked obliteration of the remaining portion of the tree and in contrast with the previous slide.

In the next slide there is evidence of the usual dilated bronchi in the bronchogram shown on the left in the presence of the atelectatic lower lobe; on the extreme right the contrast medium is in the pulmonary artery. In spite of extensive bronchiectasis because of bronchial involvement, there is a normal pulmonary arterial pattern. The middle picture shows the superimposed bronchogram in the presence of the arteriogram; there is extensive bronchial involvement in the bronchogram, with no evidence of the third and fourth order of the bronchial tree. But in the arteriogram the pattern is normal, showing all the small arterial divisions. In the next slide you see again a similar contrast. This is an excised bronchiectatic lobe, a postexcisional bronchogram on the right showing very extensive bronchiectasis. In contrast to this, however, the arteriogram in the region of the disease is normal. This explains to me the reason for the disparity between anoxia in the patient with bronchiectasis as contrasted with the patient who has more marked pulmonary tuberculosis, and still shows no evidence of oxygen desaturation. Under exercise, as Dr. Cournand pointed out yesterday, the perfusion of the lung increases to a great extent. Here is a lung that is being perfused because it has a normal pulmonary arterial system in the presence of a lung that is so damaged that it cannot be oxygenated. Under exercise the patients become more markedly desaturated as contrasted to the tuberculous patients. This explains to me also the urgency of doing operation on young patients because they are chronically anoxic, when if allowed to go on it leads to some of the difficulties of operation later in life. There is chronic anoxia, failure of development, cyanosis, clubbing of fingers, and hemoptyses.

DR. R. M. JANES, Toronto, Ontario.—I would like to say that I believe the concept which Dr. Kergin has presented with regard to management of bilateral bronchiectasis is sound, and to call your attention to the fact that in the last three years there has been no mortality. I believe his suggestion, and it was his originally, that the lesser side in some cases should be operated upon first, is also sound, in that it leaves a sufficient lung for adequate ventilation during the second stage, and anoxia during and following operation, certainly in our experience in recent years, is the greatest hazard the patient faces.

I should like particularly to call attention to the type of bronchogram developed by our department of radiology. I do not know how generally spot films are being used; spot films similar to those being used in gastrointestinal roentgenology. (Slides.) This is an ordinary posteroanterior and lateral film of the bronchogram of a normal lung. These are additional spot films, which we now employ in all cases; they are taken when it is observed under the screen that the segments it is desired to outline are filled. I think if you had time to study those you would see that they enable you to visualize every individual segment of the bronchial tree.

These have been a great comfort to us and have enabled us to approach operation upon these patients with complete certainty that we knew the total extent of the disease.

DR. WILLIAM LEES, Ann Arbor.—I should like to interject two main points, one with regard to the use of antibiotics in preoperative preparation of these patients. In Ann Arbor for a number of years we have found that the aerosol or nebulization of penicillin solution has, as Dr. Kergin mentioned, not been of too much value in preoperative reduction of foul sputum. In contrast we have used intrabronchial instillation of a solution of penicillin and have found this to be of great value. This was brought to mind by the reports in 1945 and 1946 that penicillin solution *in vitro* was somewhat more effective against certain organisms than it was *in vivo* and the bronchi in bronchiectasis are akin to test tubes. To date we have continued to use this as the method of choice. More recently we have used crystallized penicillin or the inhalation of finely divided crystals. I would like to give a word of caution here inasmuch as two patients that I have observed, and three others reported to me by the dermatologists at the University Hospital, have suffered severe ulcerative stomatitis and, therefore, I think we should be careful in recommending utilization of the micro-crystals of penicillin as a routine.

The second point I would like to mention is something which occurred to us about one year ago; that is, the difficulty which all thoracic surgeons have with the upper lobe of the "down lung" side. In the use of the lateral decubitus position with the patient placed in the Trendelenburg position, the upper lobe is the usual catch basin for all secretions, whether it be a tuberculous or nontuberculous lung. It occurred to us that perhaps we should use the reverse Trendelenburg position or, if you will, Fowler's position, inasmuch as we all know the inherent difficulty in attempting to place even a vertebrated suction tip into an upper lobe bronchus for bronchoscopic aspiration of secretions. However, even if the secretions do flow into the other side in the reverse Trendelenburg position, they will be caught by the lower lobe and one can then insert the suction tip into the individual secondary branch bronchi. This has been done in only six cases to date, and may prove to be better than the Trendelenburg position.

DR. D. H. WATERMAN, Knoxville, Tenn.—There are two or three items that I believe deserve some comment; one is the use of spot films mentioned by Dr. Jones. I heartily concur, but would also like to point out the tremendous advantage of posteroanterior stereo-bronchograms. Dr. Duane Carr, I believe, has advocated this technique for some time.

Our experience with aerosol administration of penicillin has apparently been better than that reported by other clinicians. In bronchiectatic patients we have usually combined it preoperatively with intramuscular penicillin, so possibly the true benefit is difficult to evaluate.

The third point I should like to mention regards the occasional patient who develops additional bronchiectasis following adequate excision of all demonstrably involved tissue. Very little has been written on the subject, yet occasional cases are encountered. We have had 5 in our series. Two have been diabetics, 1 of whom was a situs inversus who had, we felt, a congenital bronchiectasis. In his case we had done a left lower lobectomy, which would have corresponded, of course, to a right lower. He later developed definite bronchiectasis in the left middle and upper lobes and required excision of the remaining portion of that lung. We had another boy with right lower lobe sacular bronchiectasis with an entirely normal bronchogram of the right middle and right upper, who after right lower lobectomy developed severe bronchiectasis in the right middle lobe and the anterior inferior division of the right upper necessitating further excision. We have had 2 others in whom the left lower lobe and lingula were removed, and who subsequently developed bronchiectasis contralaterally in the right middle lobe. In another in whom the left lower lobe was removed leaving an apparently normal lingula, later lingulectomy was necessary.

In discussing the etiological factors that may be back of some of these recurrent bronchiectases, I believe we have to take into account that there may be some individuals, such as diabetics, who quite possibly have a predilection to the formation of bronchiectasis.

Associated sinus disease that has not been thoroughly controlled certainly may play a part in later development of bronchial damage. Chronic bronchitis, which always accompanies bronchiectasis, and which is not adequately treated following lobectomy may also conceivably be an etiological factor. Associated suppurative disease with its attendant difficulties, as mentioned by Dr. Lambert, may occasionally be to blame. In postoperative complications I would like to call attention to two or three things of possible etiological significance. One is the fixation of the chest wall that frequently results after lobectomy and that may be largely avoided by going in intercostally rather than by resecting a rib. In this regard, we feel that the restoration of normal chest wall function is very important following resection. Another causative factor may be the fixation and elevation of the diaphragm that often follows lower lobe lobectomy, and another may be the displacement of the remaining lobes with consequent kinking of the bronchi. Overdistention of the remaining lobes might also be a factor—we have seen bronchial dilatation occur subsequently in these overdistended emphysematous lobes. Dr. Kergin pointed out some of the measures that suggest themselves in correcting these etiological factors; certainly postoperative breathing exercises are of real importance.

We feel that this particular group I have mentioned is one that deserves our careful attention. In spite of the excellent results we are obtaining with lobectomy in bronchiectasis we must recognize that there are occasional patients who will develop further bronchiectatic changes.

DR. DERYL HART, Durham, N. C.—In repetition of something said before this Society last year, I would like to mention in the matter of control of secretions a factor which aids in oxygenation during operation and may tend to limit the spread. This is some work done by Dr. Moody on my staff in developing a method of occluding the bronchus. When he wanted to work on this problem I suggested that if he could make something to fit into the bronchus that was of the nature of a cat's-paw, to protrude small claws when he wanted them protruded and could retract them when he wanted to withdraw it without danger of scratching or irritating the trachea or bronchus, that he might be able to keep it in place. This has proved very effective in controlling the secretions from the lobe, it stays in position very satisfactorily except in the presence of extensive granulations where, in making the teeth short enough so they will not protrude through the bronchus, they may not be deep enough to take hold of very extensive granulation tissue. He can insert this into the bronchus of the lobe to be resected, air can be aspirated giving atelectasis of the lobe if it is not already totally atelectatic, and during the manipulation of freeing the lobe it prevents secretions from getting into the bronchial tree and trachea, thereby simplifying greatly the problem of the anesthetist in keeping the airways open. After the lobe is freed and just before the bronchus is clamped, the balloon is collapsed, the teeth retract, and in the same second that this takes place the bronchus is collapsed so that if secretions had not been aspirated continuously during the manipulation, that are retained in the bronchus or caught by squeezing the bronchus with the finger, the instrument is withdrawn.

I simply present this as a method of aiding in keeping the trachea free during operation and of preventing the spread of secretions to other parts of the lung.

COL. JAMES H. FORSEE, Denver.—The recording of large series of patients with bronchiectasis treated by surgical means is significant because the splendid results from surgical management are not yet thoroughly appreciated. We would like to record the experience at Fitzsimons Hospital during the past three years in the management of unilateral bronchiectasis by lobectomy in a series of 164 patients with one death. The mortality figures, however, are not alone the important figures. In the military service, in particular, we have the problem of returning patients to service. Some few years ago it was almost routine practice to retire officers and discharge enlisted personnel who had had a lobectomy for bronchiectasis. During the past eighteen months we have had only one empyema develop

following lobectomy for bronchiectasis, and it is seldom indeed that an officer is retired or an enlisted individual is discharged with a certificate of disability because of the surgical removal of one lobe for bronchiectasis.

I would particularly like to congratulate Dr. Kergin on the splendid presentation of bilateral lobectomies in bilateral disease. We have not been quite as enthusiastic, we will say, in that our number of bilateral lobectomies have been very few.

DR. LAMBERT (closing).—I would like to mention two points in closing; one is that I consider bronchiectasis a progressive disease, not in so far that it involves the bronchi, but in so far that the pulmonary tissue associated with the bronchus will be further damaged. The second point is that in this series I reported there is such a high incidence of complications and mortality that this group represents a group that are extremely ill, and also represent the cases that we did earlier in our experience with resection. Witness the fact that the early cases had almost no individual ligations and we were just beginning to do segmental resections in the later cases. Now, of course, segmental resection is almost universally employed on our service.

I feel that it is important to determine the amount of bronchiectasis present before resection is undertaken. We have not been able in some cases to appreciate exactly whether the bronchiectasis will develop following angulation of a lobe that remains or a segment that remains, or whether it was present originally. I feel that the methods of Dr. Jones and some of the other discussors, particularly the method of Dr. Gordon in the arterial injection, would be of value, although we have not tried that.

This is a very discouraging group of cases and when I summarized them I was amazed at the amount of tuberculosis that developed. I do not know exactly how to account for it. I feel that the mortality was high, but I feel that, considering the type of patient we were dealing with, that the ultimate result would have been fatal had surgery not been employed.

DR. KERGIN.—There is little that I wish to add. I am glad Dr. Adams stressed the remarkable degree of function following these very extensive resections. It seems to us that the degree of function depends not so much on the amount of lung left as upon the excellence of function of the chest wall and the diaphragm. It is because of our desire to protect that function that we have been disturbed about these serious complications following our preservation of the superior segment.

In reply to Dr. Waterman, we have been fortunate and we have not seen fresh bronchiectasis develop on the opposite side in any of this group or in our unilateral cases. We have seen bronchiectasis develop as a complication on the side of operation when there was difficulty with the lung upon which operation had been done, chiefly in maintaining a clear bronchus and in overcoming postoperative atelectasis and infection.

THORACOPLASTY

A STUDY OF FIFTY-SIX CONSECUTIVE CASES

ALFRED GOLDMAN, M.D., J. SEGAL, M.D., F.A.C.P., AND LEON H. SHERMAN, M.D.
LOS ANGELES, CALIF.

THORACOPLASTY is today a widely employed surgical procedure to arrest pulmonary tuberculous lesions. We utilize the modern thoracoplasty operation as described in 1925 and 1937 by Alexander,¹ and in 1933 by Carter² and by O'Brien.³ Our aim in performing a thoracoplasty is to bring about a selective collapse of diseased lung with a minimal interference in respiratory physiology.

The cases analyzed in this paper represent a consecutive series of thoracoplasties performed in the Los Angeles Sanatorium in the 33 month period from January, 1945, to September, 1947. This group comprises 56 cases, the latest of which is at least 6 months since its surgery. Our series is a small one, and the cases are comparatively recent. We have tried to analyze the clinical and pathologic factors involved and the early response to surgical collapse.

In the 56 cases, 170 stages of thoracoplasty were performed. Our surgical procedure is the posterolateral approach, as developed by the previously mentioned authors, in which the greater part of three ribs are usually removed at the first stage, and variable parts of two ribs at succeeding stages. Transverse processes are removed below the first rib to obliterate the costovertebral gutter. Anesthesia has generally been local infiltration with Novocain except in the first posterior stage, where nitrous oxide oxygen is given. In a selected group of cases we have found it advantageous to do a preliminary anterior stage. This group comprises the older patients and those with constitutional factors who are expected to respond poorly to the operation. It serves to test a patient's ability to tolerate surgery, especially those who fear the operation. The mild postoperative course of the anterior stage increases the patient's confidence in his ability to withstand the other stages. We have observed no untoward retention of sputum and the results have been generally good.

All of the patients were white. There were 39 men and 17 women. Ages (Fig. 1) ranged from 25 to 61 years. There were 18 per cent between 25 and 30 years, 34 per cent between 31 and 40 years, 35 per cent between 41 and 50 years, and 12.5 per cent between 51 and 61 years. The majority of our cases, 69 per cent, were in the 31 to 50 year age group. Our group is approximately 10 years older than that reported by Aufses⁴ and by Meltzer.⁵ By classification of disease, 33, or 59 per cent, of our patients had far-advanced disease, and 23, or 41 per cent, had moderately advanced disease (Fig. 2). These are average findings. Meltzer's⁵ cases give a higher degree of far-advanced disease, namely 75 per cent. The duration of disease (Fig. 3) we calculate as the time interval

From the Thoracic Surgical and the Tuberculosis Services of the Los Angeles Sanatorium.
Read at a meeting of the Trudeau Society, of Los Angeles County, Calif., May 25, 1948.
Received for publication July 1, 1948.

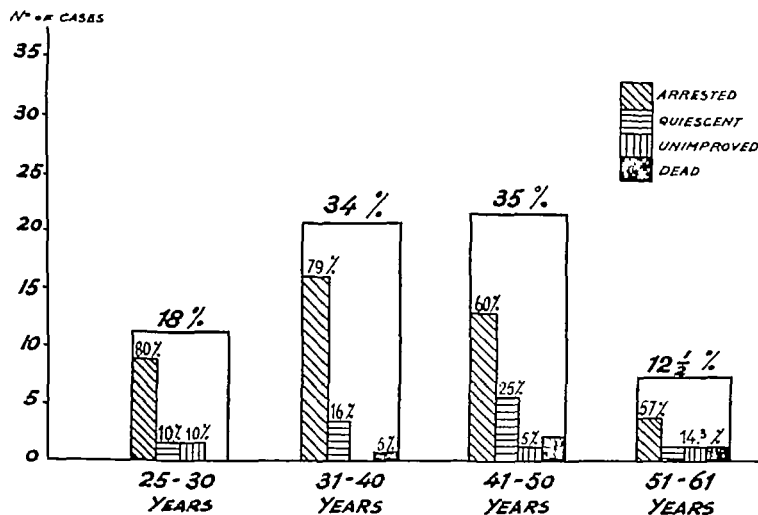


Fig. 1.—Results by age groups.

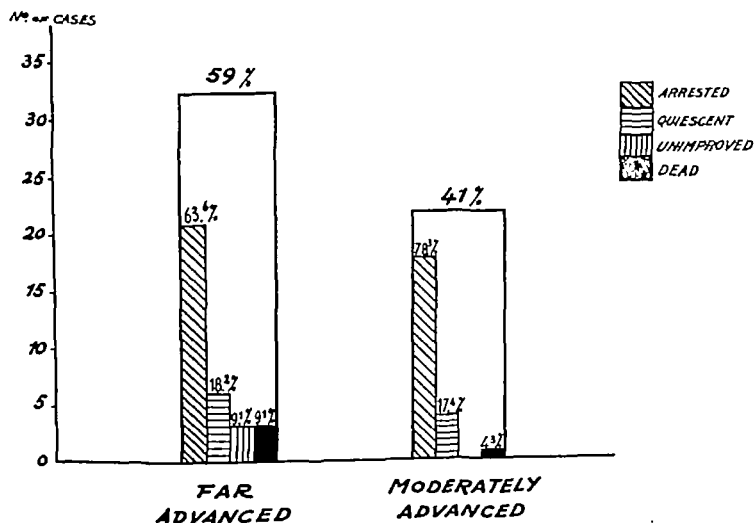


Fig. 2.—Classification of disease result.

between the diagnosis and the surgery. Forty-eight per cent, or 27 cases, had disease from 1 to 5 years, 27 per cent from 6 to 10 years, 12.5 per cent from 11 to 15 years, and 12.5 per cent from 16 to 23 years. Our group shows longer standing disease than is reported by others. Aufses,¹ out of 90 cases, gives 68.8 per cent of his cases between 1 and 5 years, 21.1 per cent between 6 and 10 years, and 10 per cent over 10 years. Meltzer,² out of 140 cases, gives 75 per cent having their disease 1 to 5 years, 20 per cent 6 to 10 years, and 5 per cent over 10 years.

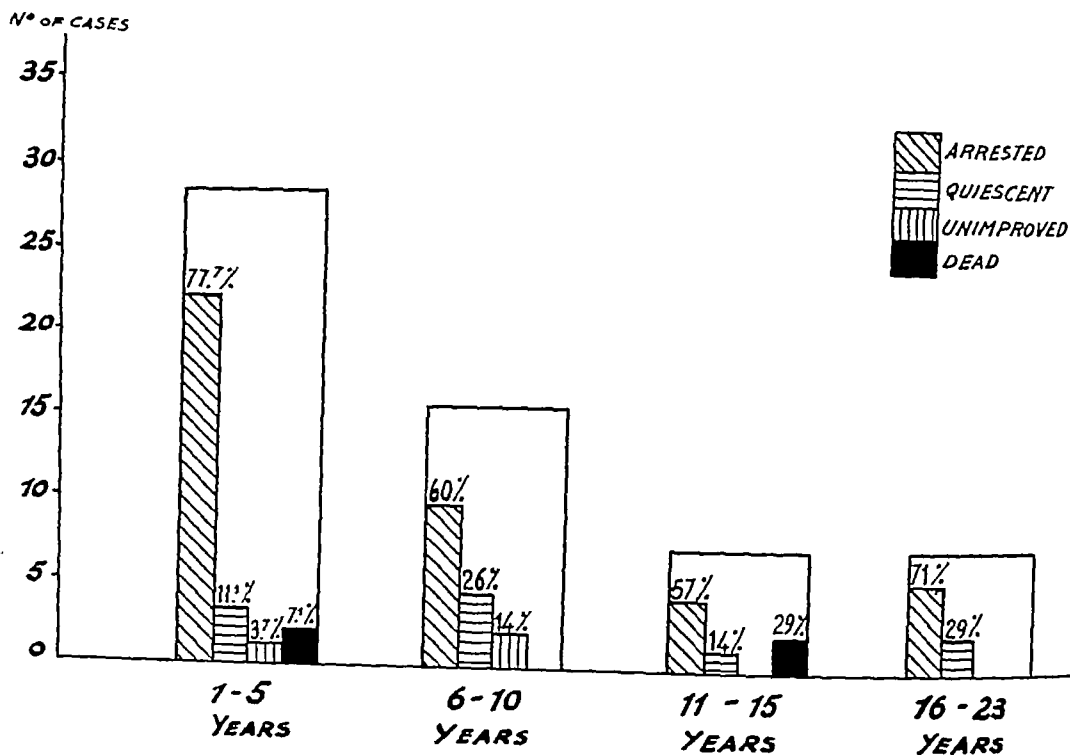


Fig. 3.—Duration of disease results.

Practically all of our patients who become candidates for thoracoplasty have electrocardiographic tracings, among other tests, to determine their cardiac status. We have been interested in finding out how those with unfavorable electrocardiographs fared following thoracoplasty. By unfavorable tracings, we are referring to those showing evidence of myocardial impairment or cor pulmonale. Fourteen such cases were approved for surgery because they appeared to be clinically suited for the operation.

RESULTS

We have tabulated the results according to the standards of the National Tuberculosis Association. By these standards, we submit the following results on 56 cases:

39 cases	70%	Arrested*
10 cases	18%	Quiescent
3 cases	5%	Unimproved
4 cases	7%	Dead

*One patient, after 19 months with negative sputum and a work capacity of 6 hours daily, recently had a positive sputum, the result of a stormy postoperative course after gastrectomy.

In studying the results of thoracoplasty according to age, we found that the groups of patients between 25 and 30 years and between 31 and 40 years gave almost the same results—about 80 per cent arrested (Fig. 1). The group over 40 years showed 60 per cent arrested, which we consider good for this age group. The surgery was borne well by them. Zuelke, Adams, and Bloch⁶ reported 68 per cent arrested among 32 patients between 40 and 65 years of age.

Within the group of far-advanced disease, 21 cases or 63.6 per cent were arrested, 6 cases or 18.2 per cent became quiescent, 3 or 9.1 per cent did not improve, and 3 patients died (Fig. 2). Among the moderately advanced disease, 18 cases or 78.3 per cent were arrested, 4 or 17.4 per cent were quiescent, and 1 patient or 4.3 per cent died. Significant we think are the results according to duration of disease (Fig. 3). Best results, as would be expected, are in the group whose disease has been present between 1 and 5 years. This group of 27 cases shows 77.7 per cent arrested. Next best to this group we were surprised to find is the one of long-standing disease, 16 to 23 years. Out of 7 cases, 71 per cent were arrested. What is more remarkable, all of the cases improved. There were no fatalities. While a few cases make percentages difficult to evaluate, we think these findings are unexpectedly good. Our results thus differ from those of Meltzer⁷ who found a decline in good results with older disease, especially after 10 years duration. Such a group showing favorable results from surgery after 16 to 23 years of tuberculosis are a naturally select group. They are the patients with "good chronics" who have themselves been able to limit their disease.

We divided the entire series into two age groups (Table I)—those up to and including 40 years of age, and those above 40 years. We found the two groups had almost the same distribution of age of disease. This is a factor that is peculiar to our material. Our sanatorium, being a national institution, draws upon patients from all parts of the country. Many have come here after having been therapeutic failures elsewhere. Some had been refused surgery for various reasons, others had not followed the cure advocated, had refused the operation in the past, and have had a change of heart several years later.

TABLE I. DURATION OF DISEASE AS DISTRIBUTED AMONG YOUNGER AND OLDER PATIENTS

DURATION OF DISEASE	1 TO 5 YEARS	6 TO 10 YEARS	11 TO 15 YEARS	16 TO 23 YEARS
Patients below 41 years of age	14	9	3	3
Patients 41 years and above	13	6	4	4

The 14 patients whose electrocardiographic tracings showed evidence of myocardial impairment were studied for the extent of surgery tolerated, the complications encountered, and the conversion results achieved (Table II). It will be noted that there is no relation between the number of ribs removed and the cardiorespiratory complications found. A total of 40 stages were performed on this group. There were four complications that could be attributed to a weakened heart, one case of cyanosis and dyspnea of three days' duration,

TABLE II. EFFECT OF THORACOPLASTY ON PATIENTS WITH ELECTROCARDIOGRAPHIC EVIDENCE OF MYOCARDIAL DAMAGE

EXTENT OF SURGERY	COMPLICATIONS	RESULTS
3-rib ant. and post. 2 stage	Cyanosis, dyspnea for 3 days following 1st stage	Arrested
4-rib ant. plus Monaldi drainage	No cardiorespiratory complications	Died (CA)
5-rib, 1 ant., 2 post. stages	Dyspnea following 2nd posterior stage	Quiescent
2 post. stages	No cardiorespiratory complications	Arrested
2 post. stages	Limited bronchiogenic spread	Arrested
6-rib, 2 post., 1 ant., revision	No cardiorespiratory complications	Arrested
1 ant., 2 post. stages	Mild decompensation following 3rd stage	Arrested
7-rib, 3 post., 1 ant. stages	No cardiorespiratory complications	Arrested
3 post. stages	No cardiorespiratory complications	Arrested
3 post., 1 ant. revision	No cardiorespiratory complications	Arrested
3 post. stages	No cardiorespiratory complications	Quiescent
3 post., 1 ant. stage	Mild degree shock after 2nd stage	Arrested
3 post. stages	No cardiorespiratory complications	Quiescent
7th rib plus revision	No cardiorespiratory complications	Arrested

another case of dyspnea of a short interval, a case of mild shock, and one of mild decompensation in a person known to have rheumatic heart disease. The only other complication was a bronchiogenic spread. The cardiorespiratory complications are 10 per cent of the 40 operations performed (Table III). We consider this a low figure. None of the complications resulted in a fatal issue, nor were the eventual results of sputum conversion affected by them. Of the 14 cases, 10 or 71.4 per cent were arrested, 3 or 21.5 per cent are quiescent, and 1 patient died of carcinoma. These results are equal to the percentages obtained for the group as a whole. From this study it therefore appears that successful surgery can be completed in cases which are clinically suited for the operation, but where the electrocardiographic tracings seem forbidding.

TABLE III. RESULTS IN THORACOPLASTY ON PATIENTS WITH ELECTROCARDIOGRAPHIC EVIDENCE OF MYOCARDIAL DAMAGE

Total number of patients.....	14
Total number of stages.....	40
Number of cardiorespiratory complications attributed to cardiac condition.....	4
Percentage of operations affected by cardiac complications	10%
<i>Conversion Results</i>	
Total cases	14
Number arrested	10 (71.4%)
Quiescent	3 (21.5%)
Dead	1 (7.1%)

VITAL CAPACITY LOSS

The cases were analyzed with reference to loss of vital capacity following thoracoplasty. The results are so unexpected that we are presenting them briefly for this reason (Table IV). It will be seen that there is a smaller average loss in the 8 rib thoracoplasty than in the 5 rib, and that one patient with a giant cavity lost 100 c.c. in a 7 rib thoracoplasty, where another lost 1,200 c.c. with the removal of only 3 ribs. We think that vital capacity measurements are unpredictable and should not be made a basis for selecting cases for surgery.

TABLE IV. VITAL CAPACITY LOSS ACCORDING TO EXTENT OF SURGERY

	3 RIB	5 RIB	6 RIB	7 RIB	8 RIB
Extremes of vital capacity loss	200-400	200-1200	200-1200	100-1200	100-800
Average loss	333	666	870	600	533

PRIMARY VS. DELAYED THORACOPLASTIES

Of the 56 cases, 13 were primary thoracoplasties and 41 were delayed thoracoplasties. We define delayed thoracoplasties as those performed following previous reversible collapse measures. Two cases were revisions of original thoracoplasties which had been done elsewhere. As we will see by Table V, primary thoracoplasties give a higher incidence of arrested cases, 77 per cent as against 68.3 per cent for the delayed group. In this matter we agree with Adams and Dufault⁷ who advocated thoracoplasty as an elective procedure where it will appear to do the most good without wasting time on measures that do not hold out much hope of success. Everyone recognizes that the majority of patients submitting to thoracoplasty have had one or more "reversible" methods of collapse applied in which the complications were often serious and the end results not so reversible as one might wish. A thickened, rigid pleura, a residual empyema pocket will interfere with satisfactory collapse. Frequently a crushed phrenic nerve fails to regenerate. Eighteen of our patients have thickened pleura, some to a marked degree. Three of these had encapsulated fluid, 12 had obliteration of the costophrenic or cardiophrenic angle from an old effusion that had complicated a pneumothorax; there were 16 paralyzed diaphragms, 11 right and 5 left; in 23 cases the trachea and mediastinum were displaced to the side of disease, in 3 cases to the contralateral side. These influences on the physiopathology of the thoracic cage did not favor a good collapse. It is surprising indeed that the modern thoracoplasty operation does give good results in the face of such obstacles.

TABLE V. A COMPARISON BETWEEN THE RESULTS IN PRIMARY THORACOPLASTIES AND IN THORACOPLASTIES FOLLOWING PREVIOUS REVERSIBLE COLLAPSE MEASURES

	NO. OF CASES	ARRESTED	QUIESCENT	UNIMPROVED	DEAD
Primary thoracoplasties	13	10 (77%)	1 (7.7%)	1 (7.7%)	1 (7.7%)
Delayed thoracoplasties	41	28 (68.3%)	8 (19.5%)	2 (4.9%)	3 (7.4%)
Revisions	2	1 (50%)	1 (50%)		
Total	56	39 (69%)	10 (18%)	3 (5%)	4 (8%)

PATHOLOGY

Twelve patients had unilateral disease (Table VI). Ten of these cases were arrested after surgery and two became quiescent. That is 100 per cent improvement in the unilateral group. Of the 44 patients with bilateral disease, 29 or 66 per cent of the cases were arrested, 8 or 18.2 per cent became quiescent, 3 or 6.8% were unimproved, and 4 or 9% of the patients died.

TABLE VI. RESULTS ACCORDING TO DISTRIBUTION OF DISEASE.

	NO. OF CASES	ARRESTED	QUIESCENT	UNIMPROVED	DEAD
Unilateral disease	12	10 (83%)	2 (17%)	—	—
Bilateral disease	44	29 (66%)	8 (18.2%)	3 (6.8%)	4 (9%)
Total	56	39 (69%)	10 (18%)	3 (5%)	4 (8%)

We have attempted to classify our patients by type of pathology to study the effect of thoracoplasty. We realize there are difficulties in any such classification. Few cases present one pathologic picture not complicated by another. We have divided them (Table VII) according to the most important problem in each case. Two-thirds of the first group, fibrotic or atelectatic lobe with cavity, were arrested. This is not remarkable since this type is expected to respond best to surgical collapse. Those with tension cavities and evidence of bronchial disease were all improved. We did not expect such good results. The group in which bronchiectasis was the main feature associated with small cavities also gave favorable results. Of the 4 giant cavities all were converted except in one patient who died of cancer before the surgery could be

TABLE VII. RESULTS ACCORDING TO TYPE OF PATHOLOGY

PATHOLOGY	NO. OF CASES	ARRESTED	QUIESCENT	UNIMPROVED	DEAD
Cavity in fibrotic or atelectatic lobe (no resisting bronchial disease)	22	14	4	1	3
Tension cavity with demonstrable bronchial disease	7	5	2		
Tuberculous bronchiectasis with cavitation	5	4	1		
Giant cavity	4	3			1
Cavitation with atelectasis of entire lung	2	1	1	1	
Caseous pneumonic disease with cavitation	13	10	2		
Pulmonary tuberculosis with mixed empyema	1			1	

TABLE VIII. GIANT CAVITIES

CASE NO.	SIZE OF CAVITY	AMOUNT OF SURGERY	RESULTS
17	5 by 11½ cm., depth from sternum to spine; occupies all right upper lobe	4-rib anterior stage, Monaldi	Dead; bronchiogenic CA with metastases
22	5½ by 9 cm., from clavicle to 4th anterior rib	4-rib anterior stage; 7 posterior ribs plus cavernostomy	Converted 6 weeks after last stage; arrested
36	7 by 9 cm., right upper lobe	3-rib anterior, Monaldi; 7-rib posterior	Converted 7 months after last stage; arrested
51	13 by 4½ cm., occupying all left upper lobe	8-rib posterior	Cavity closed with 3rd stage, converted 1 month after 3rd stage; arrested

completed. In two cases of cavity and atelectasis of the whole lung, one was arrested and one became quiescent. One patient with pulmonary tuberculosis and a mixed empyema had conversion of the sputum but is unimproved now since the wound broke down after a complete thoracoplasty. Thirteen cases of caseous pneumonic disease with cavitation are grouped together. The giant cavities occupied all or most of a lobe (Table VIII). Surgery was not completed in the first patient. In the second, closure was with a 4-stage thoracoplasty followed by a cavernostomy. In the third, closure was with the aid of a Monaldi, and the resection of 3 anterior and 7 posterior ribs. In the last case, a cavity occupying the entire lobe, closure and conversion were with an 8-rib posterior thoracoplasty.

We are pleased with our results on giant cavities. Excluding the first case that was lost to us because of a nontuberculous disease, the three remaining cases were converted. Shapiro and Muntz,² who had excellent results in their work on old-age patients, showed less favorable results with the giant cavities. Out of 8 cases, one was converted, 4 nonconverted, and 3 patients died. Meltzer³ reported 60 per cent conversions among the giant cavities in his series.

COMPLICATIONS

Of the 56 patients, 30 cases had no complications, and 25 cases had 34 complications of which 24 were surgical (Table IX). The deep wound infections involved the muscular and subcutaneous tissues. In no case were tubercle bacilli cultured. Staphylococcus was the usual organism grown. There have been no wound infections within the past year of this series. This is in part due to the use of penicillin routinely and also to employment of finer suture material. Five cases developed effusions that required aspiration; some of these were serous and some had variable amounts of blood. Two of these resulted in wound separation with delayed healing. There were two cases of shock. These responded rapidly to intravenous infusions. A transient pneumonitis disappeared in 16 days roentenographically, and may have been an autotuberculinization. A spread showed complete absorption in four months. The pleural tears were small and both were closed at the time of surgery. A moderate amount of subcutaneous emphysema followed one of them. The atelectasis followed the first stage of collapse of a giant cavity of the left upper lobe with spillover into the lower lobe. Bronchoscopy was done on the patient a few days following the first stage and the secretions were aspirated permitting aeration of that lobe. The case of cardiac decompensation was in a young man known to have rheumatic heart disease. He had a blowing systolic murmur on admission and had had an attack of auricular fibrillation before surgery. A right pneumothorax was unsuccessful and a thoracoplasty was planned. He withstood an anterior and two posterior stages well, but three weeks following the second posterior stage he had an acute episode of fibrillation with heart enlargement and some decompensation. For this reason the planned third stage had to be abandoned. This patient has since had negative sputum for the past eight months. The

Horner's syndrome followed an apicolysis. The enophthalmus disappeared in four months. There were four cases of dyspnea, one of which was known to be asthma, the others, unexplained. The tachycardia appeared twice in the same patient. The two mental reactions are included only because we think it significant that some patients react in this way following surgery. This is to be expected since mental instability is frequently encountered in most groups of tuberculous patients. The depressive stage cleared up in a few weeks. The paranoid condition was an exacerbation of a previously diagnosed schizophrenia. This cleared up a few months after surgery.

TABLE IX. COMPLICATIONS FOLLOWING SURGERY

Deep wound infection	6
Superficial wound infection	1
Excessive wound effusion	5
Wound separation	2
Ecchymosis around wound	1
Shock	2
Transient pneumonitis	1
Spread of disease	1
Pleural tear	2
Subcutaneous emphysema	1
Atelectasis	1
Cardiac decompensation	1
Horner's syndrome	1
Asthma	1
Dyspnea (unexplained)	3
Tachycardia (same patient)	2
Reaction to penicillin	1
Reactive depression	1
Paranoia	1
	34

STREPTOMYCIN

Nine patients received streptomycin therapy (Table X), 8 in connection with surgery and one 18 months following an unsuccessful thoracoplasty when tracheobronchial disease was discovered. The good results in the first case listed in Table X can be certainly credited to streptomycin. This patient had developed an acute tuberculous pneumonia on the same side for which surgery was planned. In addition she had a specific laryngitis and tracheobronchitis. She was extremely toxic, registered a temperature of 103° F., and her condition appeared critical. On a course of 2 Gm. of streptomycin daily, she made a remarkable improvement, the symptoms abated, and the lesion became stabilized. A three-stage thoracoplasty was then performed. The patient made an uneventful recovery. The dramatic change brought about in this patient under streptomycin is very impressive. It is highly probable that without streptomycin she would not have survived the fulminating disease. Certainly she would not have been made ready for surgery.

In two cases streptomycin was given as protection during collapse of cavities in patients with low vital capacities. It accomplished its purpose in both of these cases. In one of these conversion lasted for three months following

TABLE X. STREPTOMYCIN

INDICATION	DOSEAGE	RESULTS	COMPLICATIONS
Acute pneumonic spread in left upper lobe where thoracoplasty had been planned for large tension cavity; acute tuberculous, laryngitis, and tracheo-bronchitis	2 Gm. daily, 5 days of the week, for a period of 5 months	Dramatic improvement in toxic symptoms, control of fever, stabilization of lesion, surgery performed, case arrested	Dizziness
Protection for surgery in patient with low vital capacity and bilateral disease	1½ Gm. begun 10 days before surgery & continued for 3 mo.	Temporary conversion of sputum; patient died of anoxia 4 mo. after surgery	Elevation N.P.N., lowered PSP, dizziness
Protection for surgery, tension cavity; low vital capacity; contralateral extrapleural oleothorax	1½ Gm. 5 days weekly, 2½ mo.	No spread of disease, sputum conversion, arrested	Dizziness
Protection in collapse of tension cavity	2 Gm., 1 mo.	Sputum conversion 3 wk. following discontinuance of str.; arrested	Maculopapular rash
Protection for collapse of giant tension cavity	1½ Gm. 5 days weekly, 3½ mo.	No spread of disease; sputum conversion; arrested	None
Bronchogenic spread after 1st stage	2 Gm. daily, 4 mo.	Arrest of spread, stabilization of lesion; converted; arrested	Rise in N.P.N.
Protection with surgery in a patient who had shown poor resistance to disease; a contralateral spread a few months before surgery	1½ Gm. 5 days weekly, 11 wk.	No spread of disease following surgery, still pos. because of incomplete collapse of cavity; quiescent	Febrile reaction
Protection during surgery (cavernostomy for residual cavity following thoracoplasty)	2 Gm. daily for 11 days	Conversion of sputum for 1st time following cavernostomy; arrested	General urticaria, angioneurotic, edema of eye and forehead
Tracheobronchial disease of left main stem bronchus 18 months following unsuccessful thoracoplasty	1 Gm. daily	Treatment not completed; slight improvement noted	None

surgery. She died one month later, and a post-mortem showed a large cavity in the superior segment of the right lower lobe. The other patient who had a tension cavity and a borderline vital capacity, weathered the surgery without incident, had conversion, and the condition is now arrested. Two other cases had streptomycin for surgery on tension cavities. Both did well and conversion took place. A bronchiogenic spread was controlled in one case (it might have been controlled without the drug). One patient, who showed poor resistance to the disease, was given a course prior to surgery. While no spread developed the sputum was not converted either, but this must be attributed to incomplete surgical collapse of the cavity. One patient received it in connection with a cavernostomy following surgery. The sputum was converted and the disease was arrested.

Seven cases of the 9 showed some form of complication. The dizziness persisted after cessation of treatment for several weeks. None of the complications was permanent.

A study of the time relation between surgery and sputum conversion shows that results in 25 of the 40 cases that had conversion (62 per cent were made negative within one month of the surgery. Six, or 15 per cent, had conversion from one to two months later. Four or 10 per cent from 2 to 4 months, and 5 or 12½ per cent converted between 7 and 13 months. The reasons for nonconversion are listed by cases (Table XI). Bronchiectasis, thickened pleura, and tracheobronchial disease account for some failures. Spread of disease and incomplete surgical collapse for the others.

TABLE XI. REASONS FOR NONCONVERSION OF SPUTUM

CASE NO.	REASON
2	Numerous multilocular cavities below the main cavity, five weeks between each stage.
10	Spread of disease to left apex
14	Extensive thickened pleura, pleural pocket above partially collapsed lung before surgery, tracheobronchial disease
17	Died from CA before collapse could be effected
18	Failure of cavity to collapse, probably due to atelectasis of right upper lung
25	Failure of cavity to close
32	Incomplete collapse of cavity
33	Numerous cavities, bronchiectasis; delay of second stage, due to wound infection
40A	Incomplete collapse following revision
41	Tracheobronchial disease and bronchiectasis; persistent cavitation following collapse
45	Bronchial stenosis; atelectasis of left upper lobe
47	Bronchiectasis; incomplete collapse of cavity
50	Numerous calcifications in region of cavity preventing adequate collapse

DEATHS

There were 4 deaths in all.

The first death (first case of the series) was an anesthetic death.

The second patient died of anoxia a few hours after the third stage of a right thoracoplasty. Autopsy findings revealed no surgical cause of death. Collapsed cavities were found in both apices. The one in the left was collapsed by artificial pneumothorax. There was a thick pleura on the left, with bilateral

pleuritis, and old healed mitral endocarditis. It is felt that the pneumothorax involving the emphysematous left lung caused great strain on the respiratory reserve. Preoperative bronchspirometry might have called our attention to the low respiratory reserve of the contralateral lung in which case surgery would have been withheld.

The third patient died of anoxia $4\frac{1}{2}$ months following surgery. Autopsy revealed bilaterally adherent pleura, emphysema, and residual cavity in the superior segment of the right lower lobe. The cardiorespiratory reserve was too low to survive the surgery.

The fourth case was that of a giant cavity for which an anterior stage and Monaldi drainage were completed. The patient began developing pathologic fractures in the extremities. Further investigation revealed a rapidly enlarging bronchiogenic carcinoma. In the face of all this the surgical collapse of the cavity had to be abandoned.

SUMMARY

We have presented an analysis of 56 consecutive cases of thoracoplasty performed at the Los Angeles Sanatorium in a 33 month period. The material is of interest in that it represents a relatively greater proportion of old patients and those with long-standing diseases. Some decided to have surgical collapse done after refusing for many years.

In review it can be stated:

1. The younger patient has a better chance of having the disease arrested with thoracoplasty than the older one (80 per cent below 41 years of age; 60 per cent above 40 years of age).

2. The extent of disease has a direct bearing on the results of collapse. The worst group, far-advanced disease, gave 63.6 per cent arrested.

3. Where the disease is between one and five years' duration the best results may be expected. Our study shows that the group where disease has been present between 16 and 23 years did next best and the results were better than either the 6 to 10 year or 11 to 15 year groups.

4. Patients with stabilized electrocardiographic evidence of myocardial damage, but who appear to be clinically suitable for surgery, may be expected to tolerate staged thoracoplasties surprisingly well with few complications attributable to the heart condition. The conversion results are as good as for the noncardiac cases.

5. Primary thoracoplasty gives better conversion results than delayed thoracoplasty, 77 per cent against 68.3 per cent, and is to be preferred especially in the middle and older age group. The complications following "reversible" collapse procedures may jeopardize the results of a thoracoplasty.

6. A study of the results of collapse on different types of lesions leads to no conclusive findings in our series. However, the tension cavities were all improved. Of the patients with giant cavities (occupying all or most of the lobe) all whose surgery could be completed had arrested conditions.

7. Most complications were of a minor nature and have had little influence on the final results.

8. Streptomycin has been used with gratifying results. Its greatest usefulness in connection with surgery, it would appear, is to prepare patients with tracheobronchial disease and those whose surgery must be delayed because of a recent spread. In doses of 1 Gm. daily, it is well tolerated. In higher doses complications are frequent.

9. Our results out of 56 cases are 39 cases or 70 per cent arrested, 10 cases or 18 per cent quiescent, 3 cases or 5 per cent unimproved, 4 cases or 7 per cent dead.

Of the four deaths one was of a nontuberculous nature. Of the remaining three, one was an anesthetic death; the other two patients died of anoxia, one, early after surgery, the other, late (4½ months).

REFERENCES

1. Alexander, J.: New Thoracoplasty Procedure for Pulmonary Tuberculosis to Lessen Incidence of Postoperative Pneumonia, *Ann. Surg.* 81: 748-756, 1925.
Idem: The Collapse Therapy of Pulmonary Tuberculosis, Springfield, Ill., 1937, Charles C Thomas, publisher, pp. 450-516.
2. Carter, B. N.: A Technique of Thoracoplasty, *Surg., Gynec. & Obst.* 57: 353-358, 1933.
3. O'Brien, E. J.: Present Status of Thoracoplasty, Results and Technique, *J. THORACIC SURG.* 3: 159-165, 1933.
4. Aufses, A. H.: Results in 90 Consecutive Cases of Thoracoplasty for Pulmonary Tuberculosis, *J. THORACIC SURG.* 11: 98-108, 1941.
5. Meltzer, H.: Results of Thoracoplasty, *J. THORACIC SURGERY* 11: 84-94, 1941.
6. Zuelke, P. E., Adams, W. E., and Bloch, R. G.: Thoracoplasty for Pulmonary Tuberculosis Observations on 32 Patients in Older Age Group, *Am. Rev. Tuberc.* 54: 368-375, 1946.
7. Adams, R., and Dufault, P.: Surgery in Pulmonary Tuberculosis, *J. THORACIC SURG.* 11: 43-53, 1941.
8. Shapiro, R., and Muntz, C. W.: Thoracoplasty in Fifth and Sixth Decades, *M. Bull. Vet. Admin.* 18: 30-36, 1941.

CASE 3.—A 30-year-old man was sent to us by his general practitioner because of exudative pleurisy. He had dullness and suppressed vesicular breathing above the right lower lobe. The man did not feel ill. He had no fever and the sedimentation rate amounted to 10 mm. On the examination before the roentgen screen, there appeared to be only a very slight quantity of liquid; the dullness was caused by the fact that the diaphragm was a handbreadth too high and did not move.

It will perhaps not be surprising that our thoughts turned to a bronchostenosis, resulting in atelectasis and high position of the diaphragm. Bronchoscopy revealed nothing exceptional, nor did bronchography show any occlusion. It did appear, however, that the lower lobe was far too small. This was especially obvious on the lateral photograph (Fig. 3). The upward displaced diaphragm had compressed the pulmonary tissue around the basal bronchi and the lower part of the middle lobe. The upper lobe, however, was completely normal. Also the apical segment of the lower lobe revealed no changes, except in the lowermost part. With difficulty 20 c.c. of pleural liquid were aspirated and tubercle bacilli were cultivated from it. A strict rest cure was prescribed. After ten weeks neither physical nor x-ray examination revealed any anomalies. The lower lobe had completely re-expanded.



FIG. 4.

CASE 4.—A 27-year-old man had had a cough for a fortnight; he was short of breath and had a pain in the right side. In the right lower lobe there was dullness and suppressed vesicular breathing. Examination before the roentgen screen showed the diaphragm to stand a handbreadth too high and to move but little. Again there was a very small quantity of fluid containing lymphocytes. The cultures remained sterile. Reaction to Pirquet's test was weakly positive. The temperature varied between 92° and 94° F., but was normal after no

more than three days. Sedimentation rate was 36 mm. Bronchoscopy showed no anomalies. The bronchograms revealed that the pulmonary tissue around the basal bronchi had been compressed, and that there was a filling of only bare bronchi. This is apparent from the lateral x-ray film (Fig. 4). The lower lobe had collapsed (compression-atelectasis), the lower part of the middle lobe was also slightly reduced, but the upper lobe had a normal size and was in its usual place. The man remained in bed, and he rapidly recovered. After six weeks neither physical nor x-ray examination revealed any anomalies. Sedimentation rate was 2 mm. Lipiodol was introduced once more and it appeared that the lower lobe had normally re-expanded, as is visible on the lateral roentgenogram (Fig. 5).

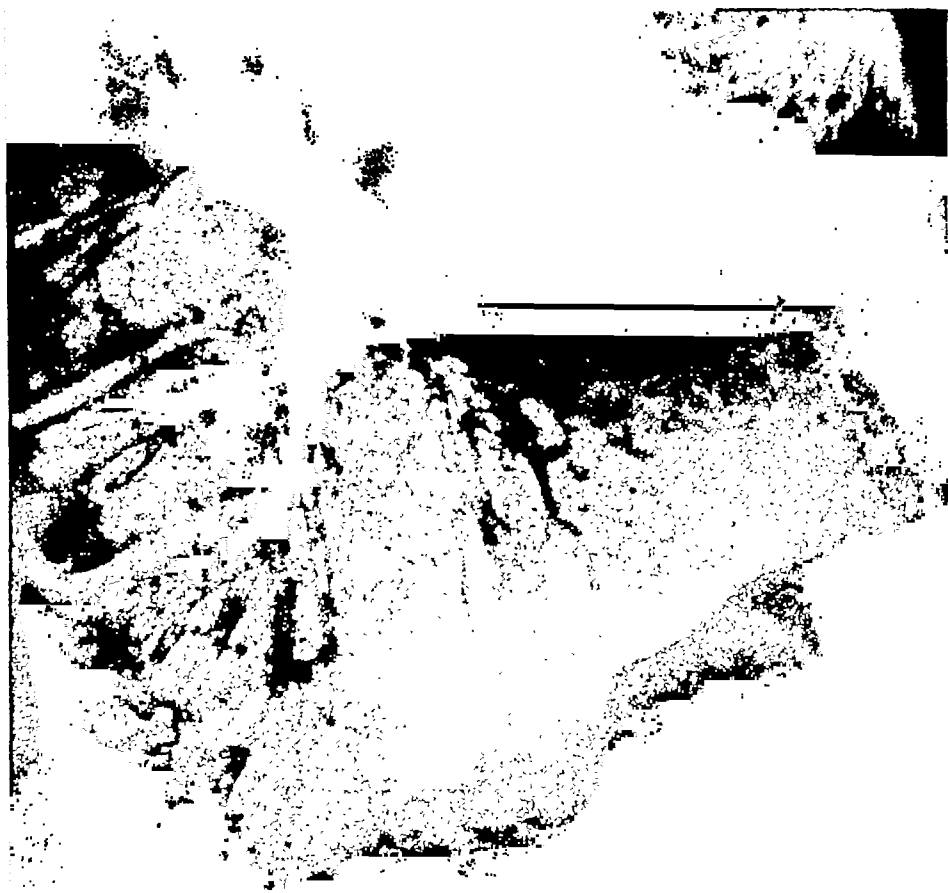


Fig. 5.

CASE 5.—Our advice was asked for a 31-year-old woman, who had been confined three months earlier. On the ninth postpartum day she had suddenly developed a pain in the right side. She had a tight feeling in the chest but did not expectorate blood, nor have a swelling of the leg. However, in view of these conditions, a pulmonary embolus must be considered. The general practitioner soon found a massive dullness. Once more the symptoms were: the diaphragm a handbreadth too high, hardly moving, and in the pleural cavity a very small quantity of fluid. In this fluid *Streptococcus viridans* was found. When bronchography was performed, the upper and middle lobe appeared normal. Of the lower lobe only the first dorsal segment was anatomically normal, the rest of the lower lobe had collapsed, due to the diaphragmatic pressure. Six weeks later the lobe had returned to normal.

The same symptoms were observed by us in one patient who had had pneumonia, and in another who had undergone a rest cure for a tuberculous pleurisy on the left side. Suddenly he developed a pain on the right side and a tight feeling in the chest: he had a high position of the diaphragm. When bronchoscopy was performed (de Graaf) no occlusion of a large bronchus was found. After two weeks the situation had cleared.

Fig. 6.



Fig. 7.

We have described patients suffering from a temporary paresis of the diaphragm. This may be caused by a process above or under the diaphragm. What causes the paresis we do not know. It is plausible to think of an inflammatory process of the diaphragm: a diaphragmatitis.

One might think that the collapse of the lower lobe resulted from the fact that the other lobes had been fixed by the pleurisy. We were, however, able to demonstrate that the same lesions are present in the patients with high position of the diaphragm after operations of the gall bladder.

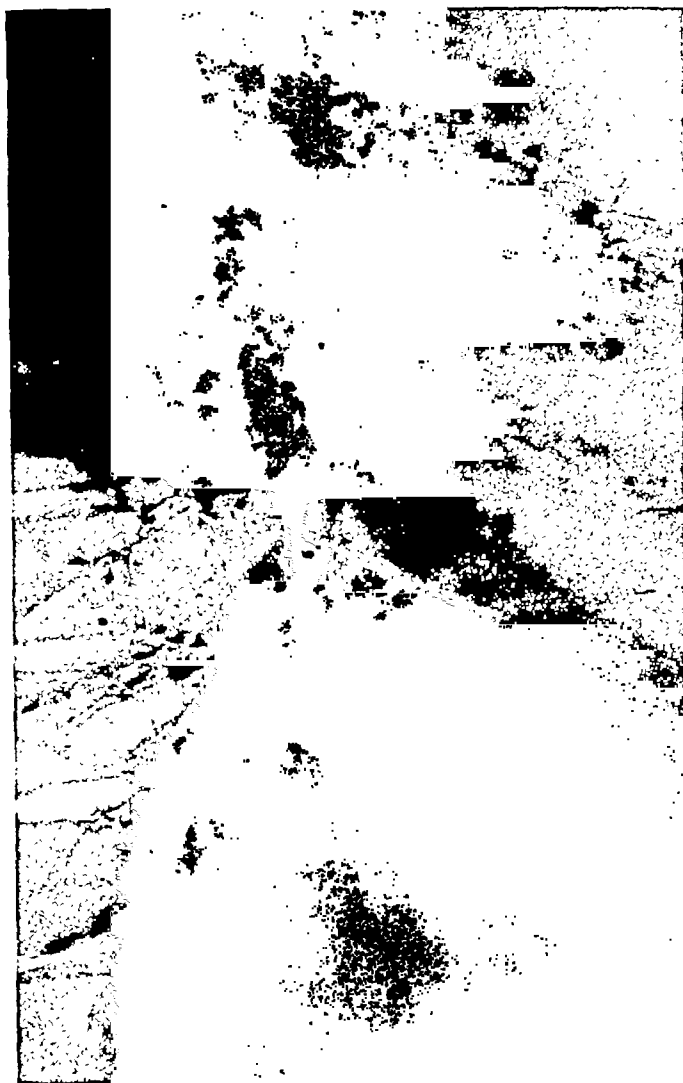


Fig. 8.

CASE 6.—A 35-year-old woman had been operated upon for cholelithiasis. She proved to have a recurrence. At the second operation no stones were found. There were, however, notably many adhesions between liver and stomach and colon, which were loosened. Three days after the operation the patient had a tight feeling in the chest and both halves of the diaphragm were too high (Fig. 6). After a few days the left diaphragm was normal again (Fig. 7), but the right half was still too high. When the woman had sufficiently recovered from the operation, a lipiodol filling was applied on the right side (de Graaf) where once again the largely collapsed right lower lobe was visible. This appears very clearly on the lateral roentgenogram (Fig. 8). It can be noted that the basal bronchi lay closely packed and were crowded toward the back. The middle lobe was not filled.

The diaphragm contains many blood vessels and lymph vessels. It is plausible to think that an inflammatory process in the lower lobe may also attack the diaphragmatic pleura, and that thus the inflammation may reach the diaphragm itself. A similar explanation might be accepted for the cases after operations on the gall bladder—an inflammation of the bed of the gall bladder reaching to the diaphragm. Owing to the operative trauma or the inflammation, fibers of the phrenic nerve may be damaged. Proof for this pathogenesis we do not have; it is a theory, no more. The possibility of a reflex has been proposed, but it is difficult to imagine that a high position of the diaphragm due to a reflex can continue to exist for a period of weeks.

It is a moot point whether a certain change in pressure works on the lung locally (Tendeloo) or spreads equally over the whole lung. This problem is of the utmost importance for pulmonary physiology; for example, is there an equal or an unequal pulmonary ventilation? It is also very important for pulmonary pathology, which in the Netherlands was pointed out especially by Tendeloo (location of pulmonary diseases). In this connection the frequently applied operative collapse therapy should be considered. Should a local or a general effect on the lung be expected from it? From our cases it appears that a lesion of the diaphragm works chiefly locally: the upper and middle lobes remain in their places, whereas the main lower part of the lower lobe and sometimes also the lower part of the middle lobe are compressed. It is clear that the reduction of the lung is achieved all but completely at the expense of the parts of the lung situated near the diaphragm.

We also often found a similar restricted influence on the lung in space filling processes in the thorax, as large tumors, pleural exudate, and thickening. The bronchograms are in this instance often almost the same, showing a compression only of the neighboring parts of the lung.

The bronchi in the compressed parts of the lung show a typical picture. The same was observed in cases of pulmonary compression due to large mediastinal tumors and in affections of the pleura. Only the larger ramifications are filled with Lipiodol; we call it a "naked filling." The bronchi are pushed together and have a curled-up appearance (Figs. 3, 4, and 8). This resembles bronchiectasis, but after expansion of the lung the bronchogram is perfectly normal (Fig. 5). It is important to know this picture.

SUMMARY

A clinical picture is described, characterized by a temporary high position of the diaphragm. Various causes are pointed out; all patients had an inflammatory process near the diaphragm, situated either above or below the diaphragm. Perhaps one should speak of a diaphragmatitis. By means of bronchography it was made clear that the high position has a very restricted influence on the lung; only the basal parts collapse, and on the upper lobe no influence can be determined. This question is probably very important for the physiology and pathology of the lung.

TRANSPLEURAL MUSCLES

LANE ALLEN, M.D.

AUGUSTA, GA.

IN THE course of a routine laboratory dissection, in a Negro man 60 years of age, a unique anomaly was found in the right pleural cavity. The anomaly consisted of two muscles lying in the oblique fissure of the right lung (Fig. 1). The medial of the two muscles arose from the sixth rib one inch from the vertebral column and inserted into the central tendon of the diaphragm just behind the caval opening. The lateral of the two muscles took origin from the sixth rib three inches from the vertebral column and inserted into the extreme lateral

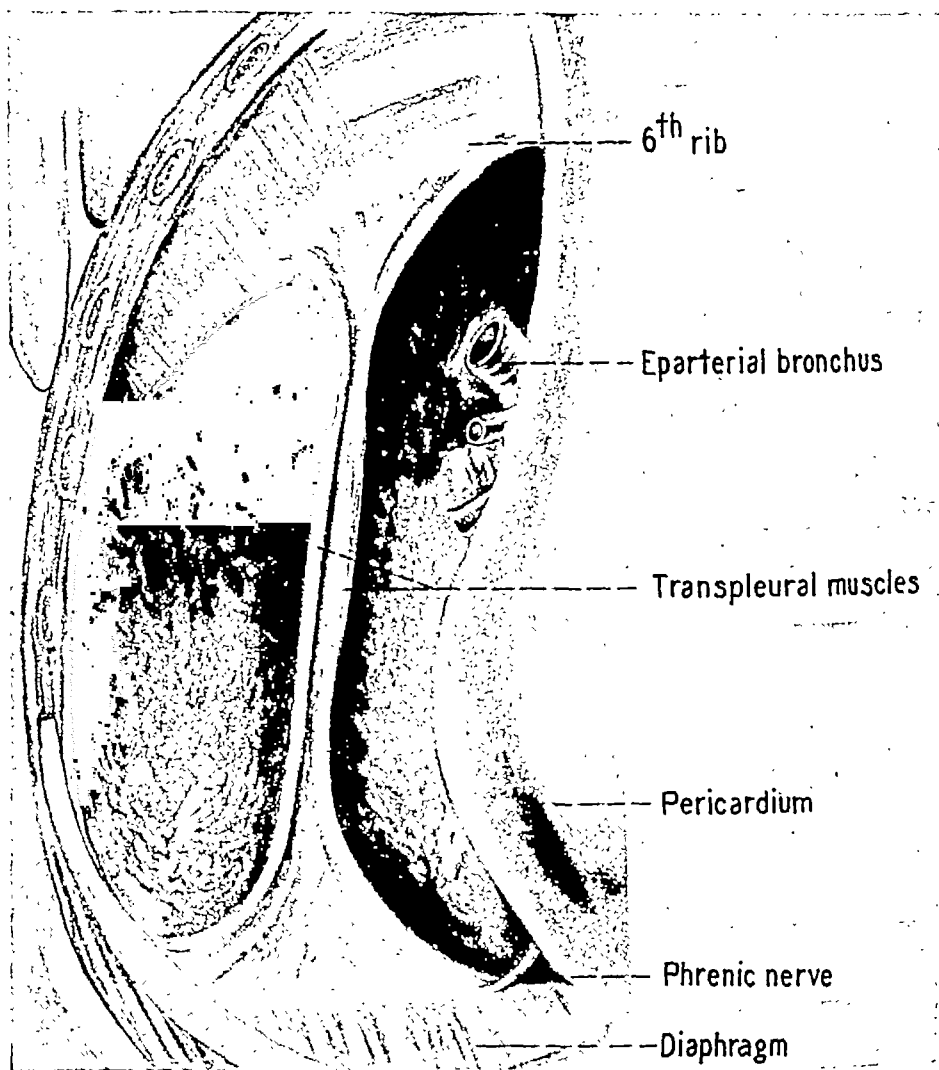


Fig. 1.—The superior and middle lobes of the right lung have been removed to show anomalous transpleural muscles in the oblique fissure.

From the Department of Gross Anatomy, University of Georgia, School of Medicine.
Received for publication July 26, 1948.

aspect of the central tendon. The muscles measured approximately six inches in length, their bellies making up about one-half the length. The bellies were slightly less than one-half inch in diameter. The tendons were thin and pearly and lay within well-defined sheaths which were condensation of loose fibrous tissue which covered the muscles and stretched between their tendons.

The origin of the nerve supply was not found, having been stripped away in the course of the student dissection. Stained section showed a preponderance of nerves in the lower portion of the muscles. This suggests that the nerve supply was phrenic rather than intercostal.

A careful search was made for associated anomalies but none were found. The possibility was considered that the fibrous sheath of the muscles was a part of an interlobar septum, but adhesions from an old pleuritic process made this impossible to determine.

It is of interest to speculate as to why similar anomalies have not been reported previously. Aside from its undoubted rarity we can only suggest that such a structure as described here might be mistaken, without close study, for an adhesion, as we first supposed it to be.

NONSPECIFIC GRANULOMATOUS (REGIONAL) ESOPHAGITIS

R. H. FRANKLIN AND SELWYN TAYLOR

LONDON, ENGLAND

THE intrathoracic causes of dysphagia in the adult usually fall into one of three main groups: cardiospasm, neoplasm, and stricture following swallowing of a corrosive. More recently interest has been taken in spasm or stricture associated with peptic ulceration of the esophagus.*

The three patients described here appear to fall into a different category and the investigations fail to show any association with tuberculosis, syphilis, or peptic ulceration, nor had any of them swallowed a corrosive. A feature common to all three patients appears to be the production of a nonspecific granulomatous lesion in the esophagus.

CASE REPORTS

CASE 1.—C. M., a 52-year-old man, had a history of progressive dysphagia extending over a period of three months. The difficulty was first noticed with solid food and later even fluids became difficult to swallow. On one occasion he noticed blood in the regurgitated material. He was examined in India by esophagoscopy and the appearance left little doubt that he was suffering from a carcinoma of the middle of the esophagus.

The patient was flown to England on May 4, 1947, and because of the almost complete dysphagia, a jejunostomy was carried out before undertaking further investigation. As soon as his condition improved, an examination by barium swallow was made and showed an obstructive lesion at the level of the root of the lung and extending over a length of about two inches. The appearance was more that of a benign than of a malignant condition. In view of this x-ray finding it was decided to carry out cautious dilatation before proceeding to esophagoscopy in the hope that the value of this examination would be increased by this measure. Dilatation was carried out with gum-elastic bougies without difficulty, and up to Size 11 was used before there was any great resistance.

A roentgenogram made on May 31, 1947, showed an irregularity of contour in the middle of the esophagus with almost complete stenosis, and the appearance was most suggestive of malignant disease (Fig. 1). Esophagoscopy was made on June 4, 1947, and the instrument passed freely to 35 cm. where a hard swelling was seen occupying one quadrant of the esophageal wall, the lumen being displaced to the opposite quadrant. The swelling bled easily but did not appear to be fungating. A biopsy was done and showed granulation tissue heavily infiltrated with pus cells, and some squamous epithelium with signs of regeneration. There was no evidence of malignancy; results of the Wassermann and Kahn tests were negative. In view of the biopsy report, treatment was continued by auto-dilatation, the patient's condition rapidly improved and he was able to resume a normal diet. A second biopsy confirmed the findings of the first.

On July 1, 1947, another x-ray examination showed no irregularity in the outline of the esophagus. This was confirmed on Sept. 6, 1947.

Further progress was uninterrupted and the patient was eating normally by June, 1948.

CASE 2.†—M. E. M., a well-developed girl 13 years of age was seen in April, 1942, complaining of increasing dysphagia. At first there was obstruction to solid food, but by the end of two months she was unable to swallow liquids and was rapidly losing weight (weight 85

From the Department of Surgery, Postgraduate Medical School of London.

Received for publication July 31, 1948.

*Allison, P. R.: Peptic Ulcer of the Oesophagus, *Thorax* 3: 20, 1948.

†We are indebted to Professor C. Crafoord for permission to include the record of patient M. E. M., who was observed by one of us (S. T.) while working at the Sabbatsberg Hospital, Stockholm, as George Hunt Travelling Scholar of Oxford University.

pounds or 38.7 kilograms). The condition appeared to arise spontaneously. On examination she was thin but healthy, and x-ray examination May 12, 1942 (Fig. 2), showed an obstructive lesion starting in the middle of the esophagus. Above the stenosed part it was of normal caliber and contour and an esophageal bougie was held up 20 cm. from the teeth. In view of the patient's condition a gastrostomy was made on June 18, 1942, and her weight rapidly rose to 91 pounds (41.2 kilograms), with a corresponding improvement in general health. In February, 1943, her condition was very satisfactory, and she was readmitted for investigation. An x-ray examination showed an obstructive lesion at the level of the aortic arch. Esophagoscopy confirmed the presence of a stricture at this level and a fine bougie could be passed no further. Gastroscopy via the stoma revealed nothing of interest and repeated attempts at all types of bouginage failed, one being followed by a brisk mediastinitis which settled with sulfapyridine. The Mantoux reaction was negative even to a dilution of 1 in 30. By 1943 the patient was unable to swallow saliva, and it was decided to explore the esophagus.



Fig. 1 (Case 1).—Roentgenogram of barium swallow.

On June 1, 1943, the chest was opened* after removal of the seventh left rib. The lung appeared normal with a few adhesions. The esophagus was mobilized from the cardia upward; its walls felt hard and infiltrated except in the lower third which felt normal. At this stage a ureteric catheter was passed up from below but would not negotiate the stricture; similarly a sound introduced via an esophagoscope above was unsuccessful. The esophagus was divided at the level of the stricture below and the distal end invaginated. The gullet was then freed by blunt dissection and brought out through an incision in the neck. The operation caused little upset and the wound healed soundly. The esophagus lying in the dressing survived for one week and then necrosis set in rapidly to within 2 cm. of the stoma. Unfortunately no specimen was sent for section until this occurred.

*Surgeon, Dr. Crafoord.

The subsequent progress of the patient was uneventful. She continued to maintain her body weight satisfactorily by means of gastrostomy feedings and was able to attend school. The stoma in the neck contracted and required dilatation, suggesting that the original pathological process was still active, although it did not involve the pharynx.

Two and one-half years later, on Jan. 14, 1946, a biopsy was taken from the esophageal stump. The section showed no mucosa in the lumen, the wall being lined by granulation tissue of dense consistency. The muscle was replaced by fibrous tissue, and there was no sign of a neoplasm.

In December, 1947, the patient was attending school and in excellent health, and the first stage in constructing an antethoracic esophagus was carried out,* using Yudin's technique.



Fig. 2 (Case 2).—Roentgenogram of barium swallow.

CASE 3.—L. C., a single woman, aged 33 years, was referred to Hammersmith Hospital on Nov. 7, 1946, with a history of dysphagia which had started on Sept. 4, 1946, and had steadily progressed until it was almost complete.

The onset of the dysphagia was sudden and occurred at the end of a long illness associated with ante-partum eclampsia. The early weeks of the pregnancy had been uneventful, and apart from the mental anxiety associated with the circumstances of the pregnancy, she remained in good health until July 21, 1946, when she had a convulsion and was admitted to a hospital where she was found to have albuminuria and hypertension. Medical induction was started but was unsuccessful, and on July 25, 1946, hysterotomy with the removal of a dead fetus was carried out. She was about thirty-two weeks pregnant at the time.

*By Dr. Crafoord.

The patient herself could remember little of what happened between July 24, 1946, and Aug. 6, 1946, but it was stated that her temperature fluctuated at this time, although there was no gross sepsis. She received no blood transfusions at any time during this illness.

It was stated that her two married sisters, who had given birth to three children, suffered from eclampsia during each pregnancy.

Her temperature gradually settled and she was considered fit for discharge on Sept. 4, 1946. At breakfast on that morning she experienced pain on swallowing and this pain was repeated during the day. Solid food was regurgitated. The symptoms became worse, but she was able to take liquids easily until the end of September.



Fig. 3 (Case 3).—Roentgenogram of barium swallow.

When the patient was referred to Hammersmith Hospital, her weight had fallen from a normal of 131 pounds (59.5 kilograms) to 87 pounds (39 kilograms). She could swallow fluids with difficulty and solids not at all. X-ray examination showed an obstructive lesion at the middle of the esophagus (Fig. 3). It was considered that further investigations were not practicable until her condition improved and a Kader-Senn gastrostomy was carried out on Nov. 8, 1946. By Nov. 22, 1946, her weight had increased to 97 pounds (43.5 kilograms), and investigations were started to confirm the nature of her obstruction. Esophagoscopy* showed an ulcer encircling the esophageal wall at a distance of 25 cm. from the alveolar margin. A portion of the ulcer was removed for biopsy and was later reported to consist of granulation tissue heavily infiltrated with polymorphonuclear leucocytes. No sign of malignancy was found. The patient had a slight anemia and occult

*By Mr. I. Griffiths.

blood was present in one out of three specimens of feces. Results from the Wassermann and Kahn tests were negative. The lungs showed no radiologic evidence of pulmonary tuberculosis, and although the sputum furnished doubtful tubercle bacilli on one occasion, this finding was not confirmed subsequently, and a guinea pig inoculation was negative.

Further radiologic studies were made through the gastrostomy opening and showed that the distal limit of the stricture was $2\frac{1}{2}$ inches below the proximal block. No lesion of the stomach or duodenum could be demonstrated, and there was no evidence of pylorospasm. At this time a drip feeding was given at night through the gastrostomy, and some of this occasionally regurgitated upward through the esophagus. The gastric acidity was normal. Small size gum-elastic bougies were passed three times a day; passage was difficult and by Jan. 4, 1947, the esophagus accepted only size No. 7.

Because of the patient's slow progress, and the continuing suspicion of malignant disease, exploration was carried out through the right side of the chest on March 11, 1947 (R. H. F.). The stricture was localized at the level of the root of the lung and was about $1\frac{1}{2}$ inches in length. Alongside the stricture was a lymph node $\frac{1}{2}$ by $\frac{1}{4}$ inch in size; both gland and stricture being firm in consistence. The esophagus above the stricture was slightly dilated. The lymph node was removed for section. The esophagus was opened just above the stricture and the incision carried downward to healthy esophagus on the distal side. The lumen of the affected portions was almost obliterated and when found would barely admit a small probe. A portion of esophagus was removed for biopsy and esophagoplasty was carried out.

Convalescence was complicated by a right-sided empyema which necessitated drainage, but the patient was able to swallow comfortably until May 28, 1947, when she experienced a "spasm" and difficulty in swallowing, which was relieved by the daily passage of a Hurst mercury bougie. She was discharged on July 11, 1947, and advised to continue the use of the mercury bougie.

Bouginage was not carried out satisfactorily by the patient, and she was readmitted on Oct. 10, 1947, with a recurrence of dysphagia. With some difficulty it was found possible to introduce a ureteric catheter, but attempts to pass anything larger than this was unsuccessful. It was decided to re-establish the gastrostomy, recover the distal end of ureteric catheter from the stomach, and withdraw the catheter upward, having attached to its tip a fine rubber tube long enough to reach outside the mouth and abdominal wound simultaneously. This was done on Nov. 4, 1947. By means of the fine rubber tubing gum-elastic bougies were guided down the esophagus. A bougie (No. 13) was passed on Nov. 19, 1947. On Dec. 18, 1947, the gastrostomy tube was removed and the gastrostomy allowed to close. Dilatation was continued, but from time to time the patient complained of painful spasms during the passage of the bougies. On March 8, 1948, the patient started to menstruate, amenorrhea having been present since onset of the illness.

The patient found the use of a bougie intolerable and resection of the affected portion of esophagus was carried out in 1948 by one of us (R. H. F.) and continuity restored by intrathoracic esophagogastrostomy. Pathologic examination showed the condition to be similar to that found in Crohn's disease. The patient has since continued in good health and has had no trouble with swallowing. By using alkalies and avoiding lying flat, she has so far not had any evidence of peptic ulceration. However, in the light of later knowledge, it would have been more desirable to have restored the continuity by using small bowel instead of stomach.

DISCUSSION

The pathologic material from these three cases presented a nonspecific granulomatous condition of the esophagus. In Case 1 (C. M.) the clinical, radiologic, and esophagosopic appearances all suggested a carcinoma of the

esophagus and if biopsy had been omitted and radiotherapy instituted, a cure by this treatment would have been claimed. The x-ray findings in the other two cases were strongly suggestive of neoplasm, even though both patients were young.

The cause of the condition is obscure; in two of the patients the onset of dysphagia was preceded by a period of mental stress. Geographically they were widely distributed: England, Sweden, and India. However, the histologic picture was similar in all three and one is tempted to compare it with regional ileitis.

The importance of this condition, which appears to be rare, is its clinical and radiologic mimicry of cancer. The diagnosis of esophageal carcinoma cannot be made without a histologic examination.

LOBECTOMY AND PNEUMONECTOMY IN THE TREATMENT OF PULMONARY TUBERCULOSIS

A SUBSEQUENT REPORT

RICHARD H. SWEET, M.D.
BOSTON, MASS.

IN MAY, 1946, a report* was made of the results of extirpative surgery applied in the treatment of 63 patients with pulmonary tuberculosis. This series was made up of 27 cases of lobectomy and 36 cases of pneumonectomy. The shortest period of follow-up in the series was one year and the longest was seven years (one case). The largest group was followed only two years (Fig. 1). At the time of this paper two years have elapsed since the last follow-up, and it is now possible to give the end results of all cases of the series after the expiration of three or more years from the date of operation. It would seem, therefore, that a subsequent report on the outcome of these cases is appropriate. This is particularly pertinent because a new factor in the form of prophylactic streptomycin therapy has been introduced since these cases were observed. It is probable that the immediate and possibly the late results will be improved because of the use of this antibiotic agent, but more time must elapse before its importance can be understood. The present series of cases, observed before the introduction of streptomycin, may assist in the establishment of a base line of the accomplishment of extirpative surgery alone, to which reference can be made in the evaluation of the assistance which streptomycin may provide in improving the results of treatment.

LOBECTOMY GROUP

The lobectomy group was comprised of 27 cases. There were 2 postoperative deaths, and by 1946 5 patients among those who survived the operation had already died as a result of the progression of the disease in the same or opposite lung. Thus there were 20 patients whose subsequent course can be studied. Of these 20 patients, 13 were apparently well at the time of the first report. Since then 6 of these have remained well continuously. Seven patients, however, developed exacerbations of the disease which required their readmission to the sanatorium for further treatment. In 3 patients the activity consisted of an exacerbation in the remainder of the lung on the operated side. Of these, 2 recovered and are now once again apparently well. The other patient is in only fair condition and is still under treatment in the hospital. One patient developed a flare-up in the opposite lung but has responded favorably to treatment and is now in an apparently well condition. One patient developed exacerbations of disease in both lungs. This patient's condition is poor at the present time and the prognosis is certainly unfavorable. Two patients developed bronchial ulcer-

Received for publication Aug. 9, 1948.

*Sweet, R. H.: J. THORACIC SURG. 15: 373-383, 1946.

ations. One of these has shown apparent complete clearing of the process. The other is slightly improved but is still in the sanatorium. These data are summarized in Table I.

Of the 20 patients still alive in 1946 after lobectomy performed one or more years previously, 7 were then suffering from manifestations of activity of the disease in the same or opposite lung. During the two years since that time 2 of these patients have improved to the point where they can be classified as apparently well. Three others remain unimproved and the other 2 have died. These facts are illustrated in Table II.

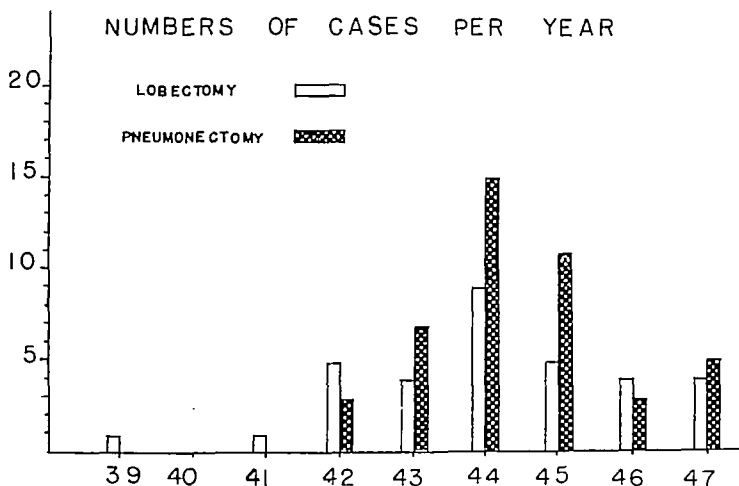


Fig. 1.

The revised end results of the original series of 27 cases are shown in Table III. Of the original 27 patients, 9 are dead. Of these, 2 died postoperatively and 7 died of late exacerbations of the tuberculosis. Of the 7 late deaths, 5 had occurred before the time of the first report and 2 occurred during the ensuing two years. Twelve are apparently well and 6 patients show some degree of activity at the present time.

PNEUMONECTOMY GROUP

The original pneumonectomy group consisted of 36 patients. Of these, 7 died postoperatively and 29 survived. But by the time the first report of results was made in 1946, 7 patients had died of the late effects of the progression of the disease. At that time also, 5 of the 22 patients still alive showed activity in the form of exacerbations in the opposite lung or of empyema on the operated side, which in the majority of cases developed late. In 2 patients these conditions

TABLE I. SUBSEQUENT COURSE OF THIRTEEN PATIENTS APPARENTLY WELL IN 1946 AFTER LOBECTOMY

CONDITION	NUMBER OF CASES	STATUS IN 1948			
		GOOD	FAIR	POOR	DIED
Remained well	6	6	0	0	0
Exacerbation in ipsilateral lung	3	2	1	0	0
Exacerbation in contralateral lung	1	1	0	0	0
Exacerbation in both lungs	1	0	0	1	0
Bronchial ulceration	2	1	1	0	0
Total	13	10	2	1	0

TABLE II. SUBSEQUENT COURSE OF SEVEN PATIENTS REPORTED IN 1946 AS ALIVE WITH ACTIVE DISEASE AFTER LOBECTOMY

STATUS IN 1948	NUMBER OF CASES
Apparently well	2
Not improved	3
Died	2
Total	7

TABLE III. END RESULTS OF LOBECTOMY FOR PULMONARY TUBERCULOSIS IN TWENTY-SEVEN CASES REPORTED IN 1946; REVISION AS OF 1948, THREE OR MORE YEARS AFTER OPERATION

Died	9
Postoperative deaths	2
Subsequent deaths	7
Alive with active disease	6
Apparently well	12
Total	27

coexisted. Seventeen patients were alive and apparently well. Table IV shows the subsequent course of these 17 patients. It will be seen from this that 12 of them remained well, but 5 developed subsequent difficulties. One sudden death from myocardial infarction, the result of coronary thrombosis, occurred. In the other 4 activation of disease in the remaining lung occurred in 2. One of these died. The other is apparently well at the present time. In one of the remaining 2 patients a small chest wall sinus developed in the incisional scar and in the other there was a flare-up of bronchial ulceration which has not yet entirely healed.

TABLE IV. SUBSEQUENT COURSE OF SEVENTEEN PATIENTS APPARENTLY WELL IN 1946 AFTER PNEUMONECTOMY

CONDITION	NUMBER OF CASES	STATUS IN 1948			
		GOOD	FAIR	POOR	DIED
Remained well	12	12	0	0	0
Activation other lung	2	1	0	0	1
Chest wall sinus	1	1	0	0	0
Bronchial disease	1	0	1	0	0
Coronary artery disease	1	0	0	0	1
Total	17	14	1	0	2

Table V shows what happened to the 5 patients who were alive but suffering from activity of the disease at the time of the first report. Two of these patients had empyema on the operated side and developed activity in the remaining lung

as well; both died. Two other patients had an empyema with fistula formation, one of the bronchus and the other into the esophagus. Both are somewhat improved as a result of thoracoplasty but not entirely well. The remaining patient succumbed to the effects of the disease in the opposite lung. Thus, of the 5 patients under discussion, none is well, 2 are alive with active disease, and 3 are dead.

TABLE V. SUBSEQUENT COURSE OF FIVE PATIENTS REPORTED IN 1946 AS ALIVE WITH ACTIVE DISEASE AFTER PNEUMONECTOMY

Apparently well	0
Somewhat improved after thoracoplasty	2
Dead	3

The revised end results of the series of 36 pneumonectomy cases are shown in Table VI. Of the original 36 patients, 19 are dead. Of these, 7 were reported previously as postoperative fatalities. Seven others had died from the late effects of the disease by the time the first report was completed. The 5 additional fatalities occurred during the ensuing two-year period of observation, making a total of 12 deaths from late activation of the disease. Three patients are alive but with evidence of activity. Fourteen of the original group are apparently well.

TABLE VI. END RESULTS OF PNEUMONECTOMY FOR PULMONARY TUBERCULOSIS IN THIRTY-SIX CASES REPORTED IN 1946; REVISION AS OF 1948, THREE OR MORE YEARS AFTER OPERATION

Died	19
Postoperative deaths	7
Subsequent deaths	12
Alive with active disease	3
Apparently well	14
Total	36

A summary of the late results of extirpative surgery as applied in the treatment of 63 patients with pulmonary tuberculosis is given in Table VII. The duration of observation varies from a minimum of three years to a maximum of seven years after the operation was performed. The majority of the cases have been followed three and four years. It is apparent from Table VII that of the 27 patients who had a lobectomy performed, 9, or 33.3 per cent, are dead; 6, or 22.2 per cent, are alive with evidences of activity of the disease, and 12, or 44.4 per cent, are apparently well. In the pneumonectomy group of 36 patients, 19, or 52.8 per cent, are dead; 3, or 8.3 per cent, are alive with active disease,

TABLE VII. END RESULTS OF EXTIRPATIVE SURGERY IN 63 CASES (THREE- TO SEVEN-YEAR FOLLOW-UP)

	LOBECTOMY		PNEUMONECTOMY		TOTAL	
	NUM- BER	PER CENT	NUM- BER	PER CENT	NUM- BER	PER CENT
Dead	9	33.3	19	52.8	28	44.4
Alive with active disease	6	22.2	3	8.3	9	14.3
Apparently well	12	44.4	14	38.9	26	41.3
Total	27		36		63	

and 14, or 38.9 per cent, are apparently well. In spite of the difference in the types of cases in each group there is a striking similarity of these figures if allowances are made for the greater postoperative mortality in the pneumonectomy group as compared with the lobectomy group (19.4 per cent versus 7.4 per cent).

DISCUSSION

Two additional years of observation of the series of patients reported in 1946 serve to bring into better focus the possible end results which can be expected after extirpative surgery in the treatment of pulmonary tuberculosis. It is very striking that of the 54 patients who survived the operation, only 19 have remained well continuously. Of these, 6 are of the lobectomy group (24 per cent of the 25 survivors) and 13 of the pneumonectomy group (45 per cent of the 29 survivors). One of the latter, however, died of heart disease. Thus, of the original 54 living patients, 35 developed subsequent reactivation of the disease after intervals of one to five or more years. Of these, 19 (76 per cent of the survivors) were in the lobectomy group and 16 (55 per cent of the survivors) were subjected to a pneumonectomy. It is obvious, therefore, that instead of the original statement that at least one-third had suffered a flare-up of activity following the surgical procedure, the present evidence suggests that approximately two-thirds (actually 75 per cent of the lobectomies) have manifested some form of late activity of persistent foci of disease. The reactivation consisted in almost all cases of flare-ups in the remaining homolateral or contralateral lung. In a few there was bronchial disease. The pneumonectomy group showed a striking incidence of late tuberculous empyema (4 cases, 2 with fistula).

Furthermore, although many of these patients improved subsequently, so that they may now be considered apparently well, 19 have died as a result of the progression of the disease (7 lobectomy and 12 pneumonectomy cases). It is more than ever apparent, therefore, that even though the major focus of disease can be excised surgically, any of the smaller remaining areas may at some future time become the site of activity of the infection. This serves to re-emphasize the importance of the immunologic aspect of tuberculosis and to stress the fact that this disease cannot be entirely eradicated by extirpative surgery applied to the lung.

It is an interesting observation that, as compared with those in the lobectomy group, the patients who had a pneumonectomy have fared rather better in so far as late results are concerned. Forty-five per cent of the survivors have remained well continuously as compared with 24 per cent of the lobectomy group. When the larger operative mortality is taken into account, however, there is relatively little difference in the over-all survival rates (Table VII).

It must not be assumed on the strength of these results that extirpative surgery should be discarded in the management of pulmonary tuberculosis. Reference to Fig. 1 will demonstrate that during the years 1946 and 1947 there were 16 resections performed, equally divided among lobectomies and pneumonectomies. The indications have been restricted somewhat, however, as a result of experience with the original series of cases reported upon first in 1946.

The indications for extirpative surgery at the present time are (1) irreversible bronchostenosis with chronic infection and secondary bronchiectatic dilatation, (2) lower lobe (or occasionally middle lobe) cavities, (3) persistent cavity after adequate thoracoplasty, and (4) possibly tuberculoma. Cases suitable for thoracoplasty are no longer considered for extirpation.

CONCLUSION

Subsequent observations of a group of patients with pulmonary tuberculosis treated by lobectomy or pneumonectomy serve to strengthen the original impression that extirpative surgery is a procedure of limited usefulness in the management of the disease. That such is the case is not surprising when proper consideration is given to the widespread dissemination of the infection which is present even at an early stage in almost all cases and to the fundamental importance of the immunologic balance between the host and the invading organism.

Whether the adjuvant prophylactic utilization of streptomycin may bring about an inhibition of the activity of the organisms sufficient to make extirpative surgery safer and more rewarding in its end results remains to be determined.

SUCCESSFUL REMOVAL OF LOWER ACCESSORY LUNG

MICHAEL DEBAKEY, M.D.,* AND J. B. AREY, M.D.,† NEW ORLEANS, LA.,
AND R. BRUNAZZI, M.D., TEXARKANA, ARK.

THE term accessory lung (aberrant or supernumerary lung) has been applied to several different types of pulmonary anomalies, of which tracheal lobes and so-called lower accessory lungs are most important. The tracheal lobe is located in the upper part of the chest and is usually connected with the trachea by an accessory bronchus, which probably represents a main and not a stem bronchus. The lower accessory lung, which is almost always situated at the base of the left lung, though it has occasionally been found below the left diaphragm, and which consists of sequestered pulmonary tissue, is not connected with the bronchial tree. It would perhaps be better termed lower accessory lobe than lower accessory lung.

The case of lower accessory lobe reported in this communication is of interest because the anomaly is a medical curiosity; in the majority of recorded cases it has been an accidental finding at necropsy, and in this instance it was successfully removed at operation.

CASE REPORT

A 7-year-old Negro girl entered Charity Hospital at New Orleans on Sept. 16, 1946, because of a "tumor" of the lung. One month before admission she had been ill for three days with a headache, nonproductive cough, and a temperature of 101° F. Her physician made a roentgenogram of the chest and told her mother that she had a "tumor" of the lung.

Examination on admission revealed an apparently normal and well-developed child. A few slightly enlarged, soft, discrete lymph nodes were found in the cervical and inguinal regions. There was a soft systolic mitral murmur. The right lung was clear, but on the left side there was impaired resonance to dullness over the lower third of the chest posteriorly. The breath sounds were considerably decreased in this region, but there were no râles and no friction rub was audible. Fluoroscopic and roentgenographic examinations of the chest revealed a well-circumscribed, oval to round cystic mass above the left diaphragm posteriorly; the mass moved with respiration; the diaphragm was mobile (Figs. 1 and 2).

The impression was that the child had either a pulmonary or a dermoid cyst, and exploration was advised. Accordingly, on Oct. 1, 1946, the thorax was explored through a posterolateral approach, under endotracheal cyclopropane, oxygen and ether anesthesia. The pleural cavity was opened by incision of the intercostal muscles in the sixth interspace. When the chest was opened, a large, smooth, somewhat rounded, brownish-gray soft mass was found lying posteriorly between the dome of the left diaphragm and the inferior surface of the left lower lobe of the lung. This mass, which measured approximately 8 cm. in diameter, was loosely adherent to the diaphragm and the lower lobe of the lung by areolar avascular tissue (Fig. 3). Elsewhere the surface, which was in contact with the thoracic wall, was smooth and glistening and resembled visceral pleura. The tumor was easily separated from the diaphragm below, and from the lower lobe of the lung above,

Received for publication Sept. 3, 1948.

*From the Department of Surgery Baylor University College of Medicine, Houston, Texas.

†From the Department of Pathology, Tulane University School of Medicine.



Fig. 1.

Fig. 2.

Fig. 1.—Anteroposterior roentgenogram of chest in case of lower accessory lung showing well-circumscribed oval to round mass lying between base of left lung and diaphragm.

Fig. 2.—Lateral roentgenogram of chest showing well-circumscribed mass lying posteriorly just above diaphragm.

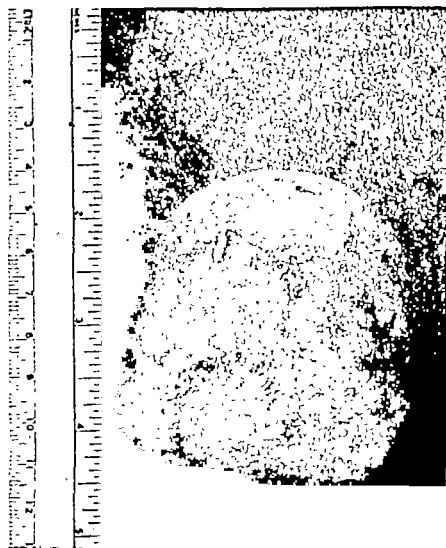


Fig. 3.—Lower accessory lung after surgical removal. Note irregularly spherical appearance and rather smooth surface.

by sharp and blunt dissection. After the mass had been freed from these structures, a small pedicle measuring 3.5 by 2 cm. was identified. The pedicle arose from the posterior mediastinum about 4 cm. above the diaphragm and well below the root of the left lung, which appeared grossly normal in all respects. The blood supply within the pedicle seemed to arise from the region between the esophagus and aorta, but the actual vascular communications were not definitely ascertained because it seemed unwise to prolong the operation by tedious dissection for this purpose. It was clear, however, that the arterial and venous supply of the tumor was systemic rather than pulmonic in origin. The pedicle was ligated and transfixed with quilting cotton sutures, and the mass was removed intact. The wound was closed in layers after the lung had been re-expanded by positive pressure.

The postoperative course was without incident and the patient was discharged from the hospital on Oct. 10, 1946.

In a follow-up report* on Nov. 17, 1947, fluoroscopic examination of the patient revealed "that the diaphragm was moving well, and that the lung appeared perfectly clear. In fact, except for the scar on the skin, it was difficult to tell that she had had any operative procedure."

Pathologic Report.†—The specimen consisted of an irregularly spherical fluctuant mass measuring approximately 8 cm. in diameter (Fig. 3). The external surface was smooth and gray except for a ragged area of brownish discoloration about 3 cm. in diameter. Minute vessels coursed over the surface. The bulk of the mass was occupied by a cyst filled with moderately thick, greenish-yellow fluid in which there were innumerable gritty particles. Concretions of similar appearance were encrusted on the lining of the cyst. In the areas free from concretions the lining of the cyst was yellowish gray and velvety. Several fibrous cords measuring up to 2 cm. in length and 0.1 cm. in diameter traversed the peripheral portion of the lumen of the cyst. The ends of these fibrous cords were attached to the cyst wall, but the cords themselves spanned small portions of the lumen of the cyst. The wall of the cyst was tough, gray, and fibrous and averaged 0.2 cm. in thickness, except at the points where the cords were attached; here it was thickened to as much as 0.5 cm. Within the thickened wall, at a point at which one of the fibrous cords was attached, there was a tiny cyst 0.1 cm. in diameter.

Sections were stained with hematoxylin and eosin, phosphotungstic acid hematoxylin, von Kossa's stain, Weigert's elastic tissue stain, and Ziehl-Neelsen's carbol fuchsin. In several areas the cyst was lined by pseudostratified columnar epithelium. Beneath the epithelium there was a distinct basement membrane five to ten microns in width. Beneath this membrane were lymphoid nodules with germinal centers, groups of mucous glands, and small numbers of elastic fibers and smooth muscle fibers. A number of circular to sausage-shaped plaques of hyaline cartilage were present beneath the lymphoid zone. The remainder of the cyst wall was made up of a thick layer of dense connective tissue fibers running roughly parallel to the lining of the cyst. Scattered through the fibrous wall were a number of nerves, large arteries, and veins. A large number of lymphoid aggregates of varying size were present throughout the connective tissue wall of the cyst; some of the larger aggregates appeared to represent actual lymph nodes.

In many areas the epithelial lining of the cyst was replaced by a thick layer of epithelioid cells throughout which were scattered foreign body giant cells and numerous large macrophages with foamy cytoplasm. Large irregular masses of calcium were incorporated in the wall of the cyst in these areas occurring predominantly in this inner epithelioid layer. The zone of epithelioid cells was surrounded by a layer of granulation tissue heavily infiltrated with small lymphocytes; this layer gradually merged with the dense connective tissue comprising the remainder of the cyst wall. The appearance suggested chronic inflammation and foreign body reaction rather than tuberculosis. No acid-fast bacilli could be demonstrated. There was pronounced intimal proliferation with narrowing or occlusion of the lumens of the arteries in the regions of extensive chronic inflammation.

*We are indebted to Dr. Norman van Wezel for the report.

†The pathologic study was supported in part by a research grant from the United States Public Health Service.

Scattered through the wall of the cyst peripheral to the cartilaginous plaques were a number of small, irregular spaces lined by cuboidal or low columnar cells. The majority of these spaces were elongated and were about 140 microns in length and 70 microns in width, but others were circular. Their contours were usually smooth, but some had scalloped irregular outlines. These spaces resembled the epithelized alveoli seen in regions of chronic pulmonary fibrosis. They were usually arranged in groups of ten or less, the individual spaces in each group being separated by septa of fibrous tissue, elastic fibers, capillaries, and groups of small lymphocytes. Additional spaces of comparable size and shape, which had no epithelial lining, were separated from one another and from adjoining epithelized spaces by delicate septa composed almost entirely of capillaries. The majority of these spaces, including those with and those without epithelial linings, were empty, but a few contained large mononuclear cells, with abundant pink stippled cytoplasm, which resembled the so-called alveolar macrophages.

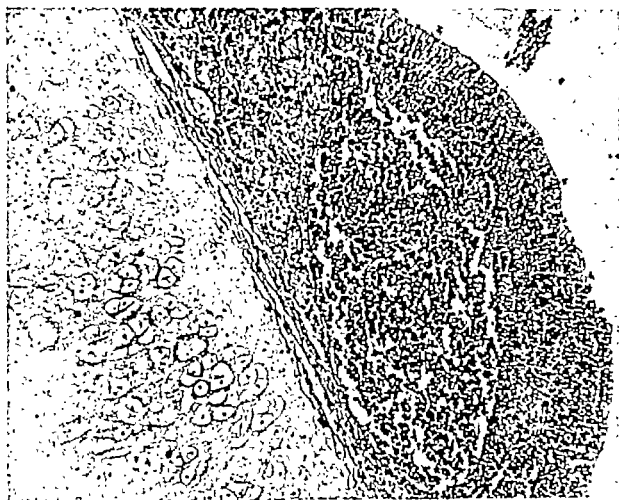


Fig. 4.—Photomicrograph of wall of cyst showing pseudostratified columnar epithelium, lymphoid aggregate, mucous glands, and hyaline cartilage.

The diagnosis of accessory lung was based on (1) the presence of a cyst lined by pseudostratified columnar epithelium and having a distinct basement membrane, mucous glands, and plaques of hyaline cartilage in its wall, and (2) the presence of epithelized and nonepithelized spaces separated by delicate vascular septa and having the appearance of pulmonary alveoli.

DISCUSSION

Lower accessory lobe is perhaps more common than the tracheal lobe, but it is still a rare condition. Since 1861 when Rektorzik and Rokitsansky each reported a case, about forty instances in human beings have been recorded in the literature.^{3-7, 9, 11, 12, 14-20, 23, 25-30, 42-46} These cases have been collected periodically and analyzed by various observers, including Bert and Fischer, Cockayne and Gladstone, Koontz, Scheidegger, Davies and Gunz, and Valle and White.

The majority of recorded cases of lower accessory lungs have been discovered at autopsy, often in infants who died at birth or soon afterward. Occasional cases have been reported in children by Quensel, von Mayenburg, and Humphry, and in adults by Vogel, Springer, and Freedlander and Gebauer. The latter authors, in 1939, successfully removed what they termed an "aberrant" lobe from a 21-year-old woman, but Valle and White, who apparently did not accept this case as authentic, in 1947, reported what they considered to be the first case of lower accessory lung discovered during the life of the patient and the sixth case in which the accessory pulmonary tissue occurred below the diaphragm. Their patient was a 9-month-old Negro boy who died, probably from shock, during operation for the repair of an associated diaphragmatic hernia. We believe our case is the first recorded of a lower accessory lung successfully removed at operation, as the case of Freedlander and Gebauer is excluded by most authors,^{7, 43} presumably because of its atypical location and character.

Although numerous theories have been advanced to explain the origin of lower accessory lungs, an entirely convincing explanation of their derivation remains to be offered. Before any explanation of their derivation can be considered adequate, certain features of lower accessory lungs must be taken into consideration, including their frequent association with defects of the diaphragm, their vascular supply, which with rare exceptions is systemic rather than pulmonary, the absence of any bronchial communication between the accessory lung and the normal lung, and the preponderance of their occurrence on the left side. In only four of the forty cases that we have been able to collect, not including our own, was the accessory lung located on the right side. The distribution in cattle, in which the anomaly has also been reported, is more nearly equal on the right and left sides.⁴¹ There are few commonly associated anomalies other than diaphragmatic hernia. The remainder of the lung is usually normal in position, size, and lobulation. The aberrant lung tissue in lower accessory lung seems to be notably free of disease or secondary infection, there being only one case¹⁷ showing tuberculous involvement in association with tuberculosis of the normal lung and two cases with bronchopneumonia.^{10, 25} This remarkable rarity of secondary infection is probably explained by the fact that few of the patients lived long enough for infection to develop and that the accessory lung tissue characteristically had neither bronchial nor vascular communication with the normally situated lung.

Two main theories have been advanced to explain the origin of lower accessory lungs, the theory of duplication and the theory of sequestration. The former was advocated by Eppinger and Schauenstein, who believed that these anomalies should be interpreted as true third lungs rather than as aberrant, sequestered lung fractions or lobes. At the present time, however, such an origin from an accessory pulmonary anlage derived from the esophagus or stomach is not generally accepted¹³ and the theory of sequestration is favored. This theory, which has been advocated by Vogel, Cockayne and Gladstone, Gruenfeld and Gray, and Davies and Gunz, attributes the aberrant pulmonary tissue to separation of a part of the primitive pulmonary anlage from the remainder

of the lung during embryonic life, with resultant formation of aberrant pulmonary tissue. Gross defects in the bronchial pattern of the normal, functional lung are occasionally found, but more frequently no such defects are described.¹²

The specimen from our case differs from many of those reported by others in the preponderance of bronchial elements and the relative scarcity of alveoli. Disproportionate development of bronchi has, however, been reported by others,^{6,7} and Freedlander and Gebauer stated that alveoli may be absent in lower accessory lungs. When alveoli are absent in a lower accessory lung, it may be impossible to distinguish it from a bronchial cyst in the mediastinum as in the third case reported by Freedlander and Gebauer. Bronchial cysts are more common in the region of the bifurcation of the trachea,^{1,2,22,24} and their walls may contain any of the tissues normally present in the trachea or bronchi.



Fig. 5.—Lower accessory lung attached to the left leaf of the diaphragm in a stillborn infant. Note the interlobular septum composed of loose connective tissue and the alveolar-like spaces with and without epithelial linings.

SUMMARY

Lower accessory lung is an extremely uncommon anomaly which probably arises during embryonic life by separation of a part of the primitive pulmonary anlage from the remainder of the lung. In the case reported in this communication the accessory lung was successfully removed at operation.

ADDENDUM

Since the completion of this paper we have observed an additional example of a lower accessory lung. This occurred in a stillborn fetus weighing 4,220 grams whose death was apparently the result of rupture of a subcapsular hematoma of the liver.

AN ARTIFICIAL AORTIC VALVE

PRELIMINARY REPORT

J. MOORE CAMPBELL, M.D., F.A.C.S.* (BY INVITATION)

OKLAHOMA CITY, OKLA.

THE problem of restoring or improving the physical efficiency of incompetent cardiac valves has received little attention in surgical research, and there has been no report in the literature of any attempt to make a complete cardiac valve replacement.

In 1914, Alexis Carrel¹ reviewed his technique by which operations on the pulmonary and aortic orifices of the heart could be performed in dogs with some degree of safety. In one experiment he was able to cut the right pulmonary valve cusp with scissors and unite the laceration with fine silk suture. When the animal survived and retained a slight diastolic murmur, he reasoned and subsequently showed that "the bottom of the valve did not unite and a certain amount of insufficiency persisted."²

In 1938, Gordon Murray³ resected with a valvulotome a portion of the lateral cusp of the mitral valve in dogs after the method of Cutler and Beck. He then took a segment of external jugular vein turned inside out, and threaded it through the anterior heart wall across the cavity of the heart to emerge on the posterior surface of the left ventricle. Anchored with slight tension, it made a free graft that bulged with systole into the opening left by the resected portion of valve. Cyanosis and cardiac dilatation were promptly controlled, and two dogs lived past ten days, remaining without signs of heart failure although they retained soft, blowing systolic murmurs.

The artificial aortic valve that forms the basis of this report is one of many valve patterns that have been fabricated from several types of plastic material. In 1937, Hufnagel⁴ showed that a cylinder of methyl methacrylate approximately 4 cm. in length, 1 mm. in thickness, and with a diameter that was approximately the internal diameter of a canine aorta, could replace an excised segment of aorta, and that when several animals were sacrificed after six months there was found little tissue reaction at the junction of the intubation anastomosis and no mural thrombi were present.

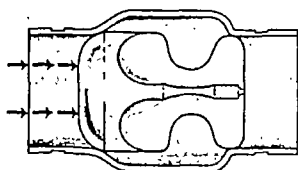
Using the same methyl methacrylate, a tube with a central chamber was machined on a lathe (Fig. 1). Into the chamber was placed a moving valve machined and carved from the plastic. The length of the tube and contained valve is about 2.5 cm., thickness of the walls a little over 1 mm., and its over-all diameter about 1.2 cm. at the ends. This particular valve model opens and closes easily and quickly, and only 3 cm. water pressure is required to seat the valve (Fig. 2). The entire prosthesis is carefully polished on all surfaces.

Read at the Twenty-eighth Annual Meeting of The American Association for Thoracic Surgery, Quebec, Canada, May 31, June 1, 2, 1948.

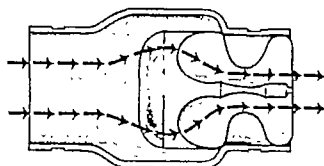
Part of financial support for this research has been granted by the United States Public Health Service, National Heart Institute, Bethesda, Md.

*Thoracic Surgeon, University of Oklahoma Hospitals, Instructor in Surgery, School of Medicine, University of Oklahoma.

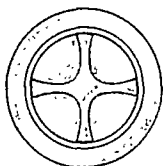
To produce aortic regurgitation in dogs, under Nembutal anesthesia the right carotid artery is exposed through a small incision in the anterior neck, and a semisharp stylet passed through the artery and aorta to aortic valve leaflets. The leaflets are deftly traumatized and aortic regurgitation is produced because of the thoroughly lacerated aortic valves. The trachea is then intubated and



Position in Diastole



Position in Systole



*End View, Distal Orifice
Artificial Aortic Valve Design*

Fig. 1.—Diagram of artificial aortic valve.

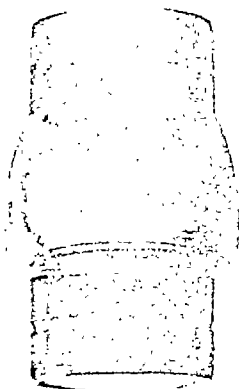
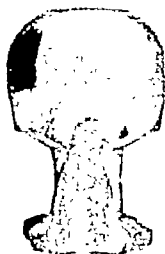


Fig. 2.—Component parts of artificial valve.

positive pressure respiration is begun. Next, a left anterolateral incision is made in the left fourth interspace, and the fourth costal cartilage cut. After packing away the left lung with moist gauze, and after all aortic branching vessels from the left subclavian to the fifth pair of intercostals are doubly ligated and divided, the aortic arch and descending thoracic aorta are thus mobilized. When completely free, the aorta is clamped at its proximal and distal dissected points, isolating from blood flow a segment of aorta about 10 cm. in length. A longitudinal opening now is cut in the vessel, the artificial valve inserted, the aortic

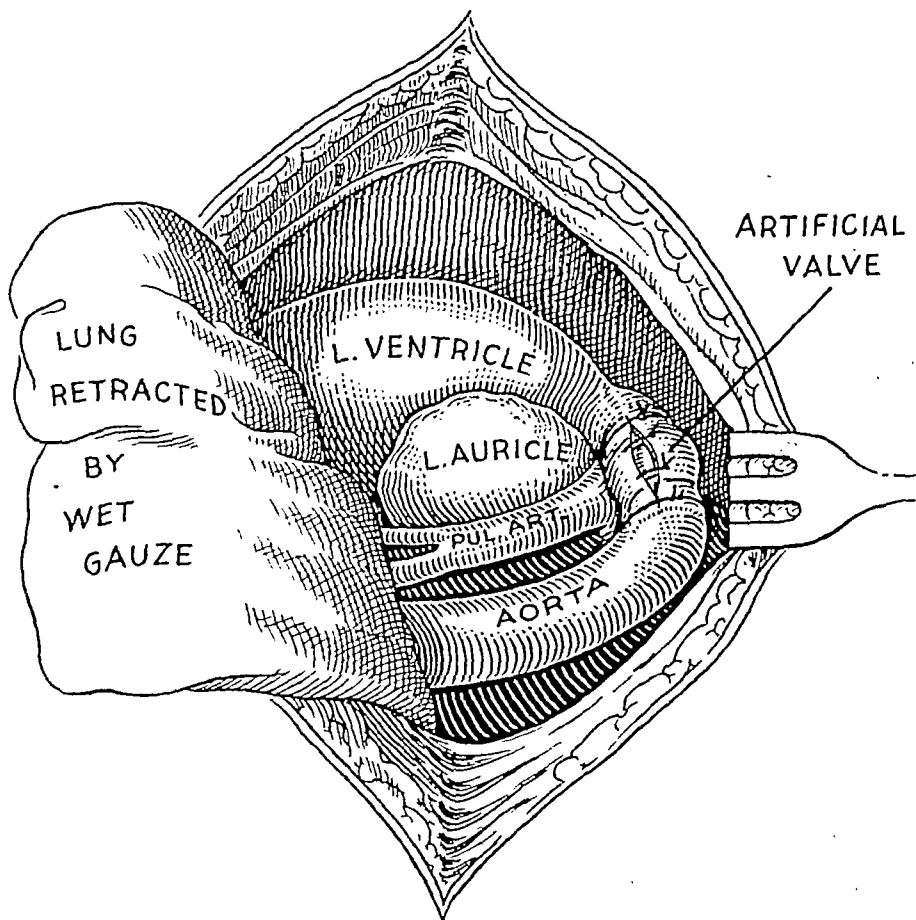


Fig. 3.—Position of artificial aortic valve. Exposure through fourth left interspace.

segment filled with citrate solution, and the valve anchored in place by means of No. 4 braided silk ligatures circling the aorta over each end of the valve chamber. The distal clamp on the aorta now is opened, then slowly the proximal one. The wound is closed with interrupted cotton sutures, and the collapsed lung re-expanded by aspirating the chest. The time interval during which the aorta is clamped off varies from $2\frac{3}{4}$ minutes to $4\frac{1}{2}$ minutes. It also has been possible to close the opening in the aorta with fine silk sutures, release the encircling ligatures, move the valve upward across the aortic arch, and anchor it much closer to the aortic leaflets than shown in Fig. 3.

OBSERVATIONS

Kymographic recordings are made with a tambour connected to a femoral artery. After the aortic valves were lacerated, the normal pressure changed to one of aortic regurgitation. The systolic pressure rose, the diastolic pressure fell, and the resultant pulse pressure markedly increased within a matter of thirty seconds to five minutes. After placing the valve in the aortic arch, recordings of blood pressure approximated the dog's original pressure, although the shape of the curve varied from normal, due in part to the snapping action of the

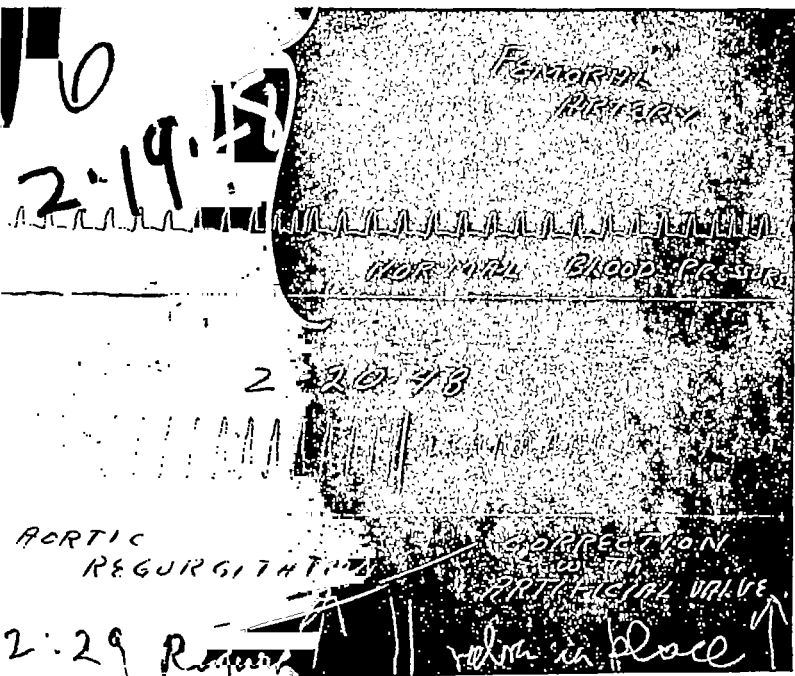


Fig. 4.—Femoral artery tracing. Normal blood pressure, aortic regurgitation, and corrected pressure with valve in place.

valve (Fig. 4). One tracing clearly showed that when open, minimal obstruction to aortic output was made by the valve or by the plastic chamber functionally replacing the aorta at its site.

A phonocardiographic record was kept of each dog. The normal heart sounds are replaced with the murmur of aortic regurgitation after tearing the aortic leaflets, and this murmur is replaced or overshadowed by the sound of the artificial valve (Fig. 5). Whether or not the valve is functioning can be easily

determined with a stethoscope, for when opening and closing it makes a sound resembling the ticking of an alarm clock. For some acoustical reason, probably the nearness of the valve prosthesis to the trachea, the ticking can easily be heard through the mouth of the dog when the jaws are held open, making a simple determination of whether the valve is in action and working from day to day.

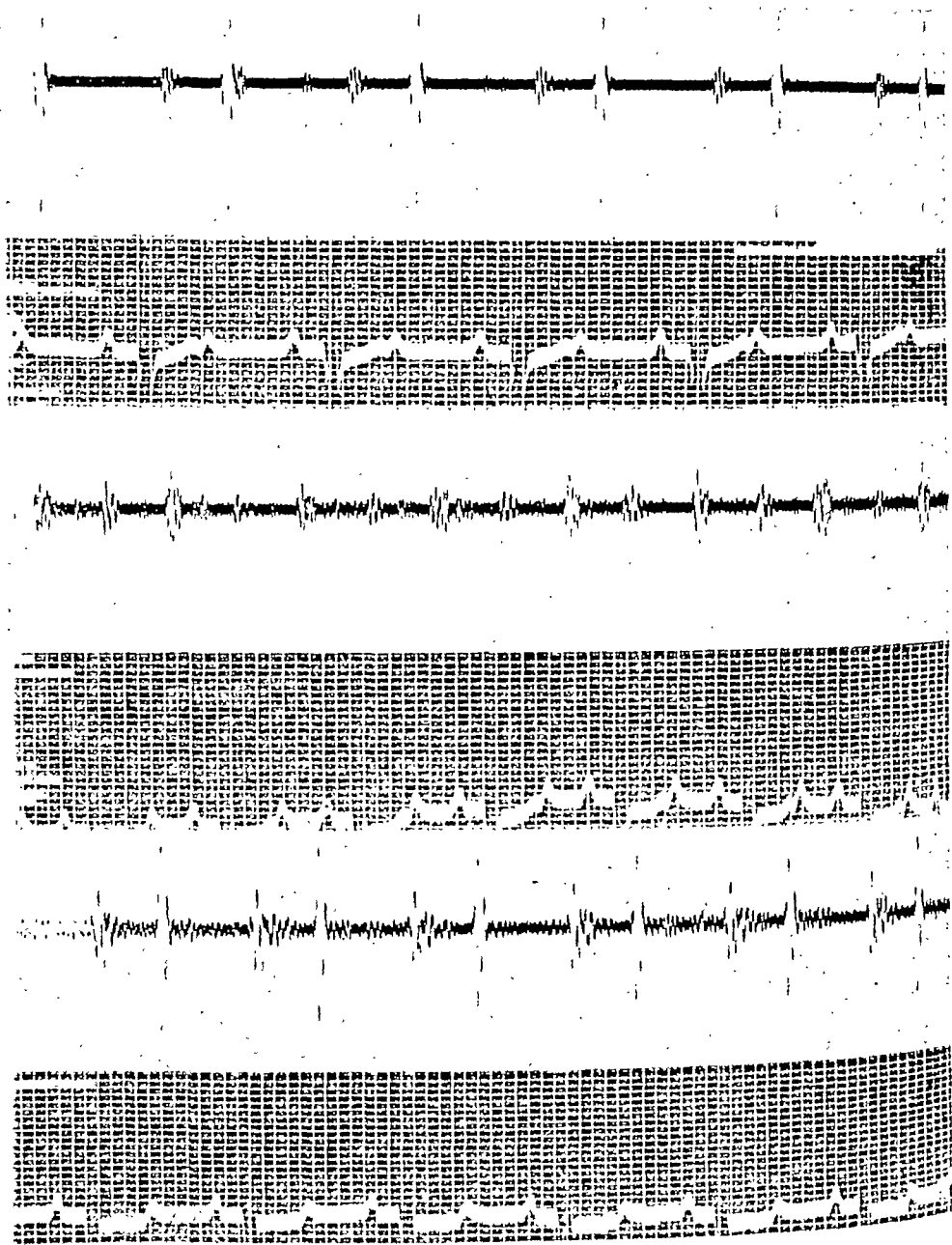


Fig. 5.—Phonocardiograph tracing of (1) normal aortic sounds, (2) aortic regurgitation, and (3) valve sounds, absence of aortic regurgitation.

With careful manipulation of the aorta, clotting of the valve at the site of its insertion has not been a problem. If, however, any traumatic ulceration of the aorta is produced while inserting the valve, thrombi are likely to develop in the

denuded areas. Anticoagulant drugs were not used in the group of dogs here reported.

In a series of five dogs in which this model valve has been employed, an elderly dog, weighing 20 kilograms, developed ventricular fibrillation twice while the aorta was clamped off, retained an arrhythmia that could not be controlled, and expired two hours postoperatively. One dog had a fatal hemorrhage on the fourth day, and the distal ligature about the valve was found to have cut through the aortic wall. Two dogs developed thrombi in the region of the lacerated aortic leaflets which dislodged on the third and fifth days and formed a plug in the valve that held it in the open position. Two dogs autopsied on the sixth and seventh postoperative days showed functioning valves, minimal tissue reaction, no evidence of thrombosis or technical difficulty. All of the dogs except the first one of this group were physically active from the first postoperative day. The ultimate survival is to be established by a series of dogs now under study and will be reported at a later date.

Although much remains to be done in these experiments, this report is made because it has been found possible to place an artificial valve in the circulatory system made of plastic substance and not autogenous tissue. With further improvement and study, it may be possible in the future to develop a valve prosthesis that may relieve aortic regurgitation in human beings, since it has been shown by these acute experiments that methyl methacrylate valves are tolerated in arterial blood flow and do not cause thrombosis as might be expected.

SUMMARY

1. Valves made of methyl methacrylate and placed within the aorta distal to lacerated aortic leaflets have been observed to function as a satisfactory correction of aortic regurgitation.
2. The valves have functioned up to seven days in this group of experiments.
3. Ultimate survival time is under study.

REFERENCES

1. Carrel, Alexis: Experimental Operation on the Orifices of the Heart, *Ann. Surg.* 60: 1, 1914.
2. Carrel, Alexis: Experimental Operations on the Sigmoid Valves of the Pulmonary Artery, *J. Exper. Med.* 20: 9-18, 1914.
3. Murray, Gordon D. W.: Reconstruction of the Valves of the Heart, *Canad. M. A. J.* 38: 317-319, 1938.
4. Hufnagel, Charles A.: Permanent Intubation of the Thoracic Aorta, *Arch. Surg.* 64: 382-389, 1947.

DISCUSSION

DR. JOHN H. GIBBON, JR., Philadelphia.—Intracardiac surgery can be performed best by clamping both superior and inferior vena cava, and then operating in the open heart under direct vision. That is possible at the present time only for very brief periods of time. I think perhaps in the future if an extracorporeal circulation can be devised which will carry the circulation it may be possible to have the heart open and to carry out operations on septa and valves under direct vision. At the present time we are limited to short periods of about four minutes. Recently, Dr. Templeton in our laboratory has been able to clamp the vena cava, to open the right auricle, and under direct vision to excise one of the cusps of the tricuspid valve. He has replaced that defect in the tricuspid valve with a graft of vein or pericardium. The grafts of pericardium have been most successful. There are now five dogs living from one to five months after operation; two

of these show no murmur at all. We have had control dogs with cusps excised who show a definite murmur confirmed by phonocardiograms. I hope Dr. Templeton* may say a word about the technique.

DR. CLARENCE CRAFOORD, Stockholm.—I think that Dr. Templeton should be congratulated on the result of these experiments. The question that Dr. Gibbon raises is important for the further development of cardiac surgery. To develop a device for extracardiac circulation and extrapulmonary oxygenation in my opinion must be considered as essential for intracardiac operations. To be independent of the active breathing by performing controlled respiration I consider is now proved to be one of the prerequisites for major intrathoracic surgery. To make oneself independent of the cardiac action for the circulation I am certain will be as essential for intracardiac surgery. In my experimental department we have been working with a device that is quite different from the one Dr. Gibbon has constructed. We have now come so far that we can take over the circulation of the brain in a full-grown dog for as long as one and one-half hours, and then remove the artificial circulation and oxygenation machine and let the heart and the lungs take over their function again with surviving normal dogs.

In these experiments we introduced a cannula into the brachiocephalic artery, which provides the brain with blood, close to the aortic arch, after clamping this artery between the cannula and the arch. We clamped the inferior and superior vena cava, and tied the azygos vein. By this means the brain was adequately perfused during the experiment. The heart was bloodless because of the clamping of the cava. From my experience in cross clamping the aorta in a series of about forty cases of patent ductus I am convinced that if only the central nervous system is adequately perfused there will be a time of between twenty and thirty minutes for operations within an opened bloodless heart.

**Editor's Note:* Dr. Templeton's discussion was not returned by him. For that reason it must be omitted here.

SODIUM TETRADECYL SULFATE, AZOCHLORAMIDE, AND SULFANILAMIDE MIXTURE IN THE PREVENTION OF EMPYEMA FOLLOWING PNEUMONECTOMY WITH CONTAMINATION

H. L. RIGDON, M.D., AND O. C. BRANTIGAN, M.D.
BALTIMORE, MD.

THE empyema that follows contamination of the pleural cavity after pneumonectomy for suppurative pulmonary disease constitutes one of the hazards in thoracic surgery. The lung to be removed is frequently adherent to the parietal pleura by adhesions formed by the underlying pulmonary pathologic conditions. In freeing an adherent lung from the chest wall, even though dissection is carried out in the extrapleural plane, a peripheral abscess is ruptured occasionally, and pus is spilled into the pleural cavity. A rupture into the cavity is more likely to occur when pulmonary resections are being done for pyogenic lung abscess and carcinoma complicated by a peripheral abscess; there is less likelihood of this rupture in resecting a lung for pulmonary tuberculosis or bronchiectasis. After division of the main bronchus, occasionally the clamp may slip from the bronchial stump, and infected bronchial secretions spilled into the pleural cavity may contain the organisms that instigate empyema. Empyema also may follow pneumonectomy where gross contamination is not apparent.

The opening of the bronchial stump with the resulting bronchopleural fistula is becoming a less common cause of empyema. However, when this type of empyema does occur, the infected pleural cavity is open to the outside through the tracheobronchial tree. This empyema is different from that caused by contamination of the pleural cavity at the time of operation when the bronchial stump remains closed. Drainage of the pleural cavity following total extirpations of the lung is almost certain to be followed by empyema if the drainage tube is allowed to remain longer than seventy-two hours.

Once empyema becomes established it is practically impossible to cure it by chemotherapy. Repeated aspirations and injections of chemotherapeutic and antibiotic agents usually will control the infection, but for a permanent cure the pleural cavity must be obliterated. This can be accomplished by drainage, and then allowing the space to become obliterated by elevation of the diaphragm, retraction of the interspaces, and overdistention of the remaining lung with a shift of the mediastinum to the affected side. Usually it is necessary to do an extensive thoracoplasty to accomplish the desired result. Strieder² reported that in his experience empyema promptly recognized and treated by extensive rib resection and open drainage is accompanied by an 85 per cent recovery rate. Temporizing measures, such as aspiration or closed drainage, result in higher mortality.

From the Department of Surgery, School of Medicine, University of Maryland.
Received for publication Sept. 23, 1948.

of these show no murmur at all. We have had control dogs with cusps excised who show a definite murmur confirmed by phonocardiograms. I hope Dr. Templeton* may say a word about the technique.

DR. CLARENCE CRAFOORD, Stockholm.—I think that Dr. Templeton should be congratulated on the result of these experiments. The question that Dr. Gibbon raises is important for the further development of cardiac surgery. To develop a device for extracardiac circulation and extrapulmonary oxygenation in my opinion must be considered as essential for intracardiac operations. To be independent of the active breathing by performing controlled respiration I consider is now proved to be one of the prerequisites for major intrathoracic surgery. To make oneself independent of the cardiac action for the circulation I am certain will be as essential for intracardiac surgery. In my experimental department we have been working with a device that is quite different from the one Dr. Gibbon has constructed. We have now come so far that we can take over the circulation of the brain in a full-grown dog for as long as one and one-half hours, and then remove the artificial circulation and oxygenation machine and let the heart and the lungs take over their function again with surviving normal dogs.

In these experiments we introduced a cannula into the brachiocephalic artery, which provides the brain with blood, close to the aortic arch, after clamping this artery between the cannula and the arch. We clamped the inferior and superior vena cava, and tied the azygos vein. By this means the brain was adequately perfused during the experiment. The heart was bloodless because of the clamping of the cava. From my experience in cross clamping the aorta in a series of about forty cases of patent ductus I am convinced that if only the central nervous system is adequately perfused there will be a time of between twenty and thirty minutes for operations within an opened bloodless heart.

*Editor's Note: Dr. Templeton's discussion was not returned by him. For that reason it must be omitted here.

tions. Petroff and Schain⁸ found that it was most effective when used in combination with azochloramide. Neter⁷ reported that the combined activity of sulfanilamide and azochloramide exerted a greater bactericidal action than when either was used separately. This suggested the possibility of combating the three drugs, azochloramide, sodium tetradeceyl sulfate, and sulfanilamide.

When the pleural cavity is contaminated at the time of pneumonectomy, essentially the same procedure is carried out now as previously described.⁴ Approximately 500 c.c. of the mixture containing azochloramide, 1:3,300, sodium tetradeceyl sulfate, 1:500, and 5 Gm. of sulfanilamide are placed in the pleural cavity. The sulfanilamide crystals are inserted in the pleural cavity and then the solution of azochloramide and sodium tetradeceyl sulfate is added. The chest is closed tightly without drainage. The intrapleural pressure is reduced to about 1 cm. of mercury negative pressure after the chest is closed. On three consecutive days after operation or until three consecutive negative pleural cultures are obtained, the pleural cavity is aspirated and the same mixture of three drugs is instilled. The quantity instilled depends upon many factors, including mechanical difficulty in aspirating and the necessity of leaving the pleural cavity under negative pressure after the aspiration and instillation. An endeavor is made to instill at least 200 c.c. and rarely more than 500 c.c. of the mixture at each treatment.

Including the first three cases reported, there have now occurred fourteen instances of grossly contaminated pleural spaces after pneumonectomy. These are consecutive cases and all patients have been treated by the method described. All have escaped infection or empyema, except the sixth one treated. This patient presented certain peculiarities that seem to warrant a detailed report.

CASE REPORT

A. S., a 40-year-old white woman, was admitted to the University Hospital on Dec. 9, 1947. In June, 1946, this patient had been diagnosed as having pulmonary tuberculosis. The third stage of a relaxing thoracoplasty was completed in April, 1947. Following this operation the tuberculosis was apparently arrested, and the patient was free of symptoms until several months prior to admission, when she developed a cough productive of a moderately large amount of sputum and a low grade fever. A bronchogram showed extensive bronchiectasis throughout the right lung under the thoracoplasty. Examination of the sputum and morning fasting gastric contents revealed no evidence of tubercle bacilli. On auscultation there was definite bronchial spasm bilaterally. The patient was moderately dyspneic. Pneumonectomy on the right side was advised and accepted. In preparation for the operation the administration of aerosol penicillin and streptomycin was prescribed. The patient became so dyspneic from bronchial spasm that she almost lost her life. Her reaction was almost as violent when the same antibiotics were injected. She was also found to be sensitive to the sulfonamides. On Dec. 29, 1947, a pneumonectomy was performed on the right side. The lung was freed from the chest wall with difficulty, and during manipulation it was noticed that there was spillage of a small amount of pus. Prior to closure of the chest the usual azochloramide, sodium tetradeceyl sulfate, and sulfanilamide mixture was used. The regular three-day routine of aspiration and injection was carried out with the same mixture. Sulfanilamide was administered in spite of the previous reactions to it. In view of the danger of infection an antihistamin drug was given, followed by penicillin in streptomycin, but none of these could be tolerated. The culture taken at operation proved positive for hemolytic *Staphylococcus albus* and gamma *Streptococcus*. A culture of the pleural fluid on the first

postoperative day showed no bacteria. The temperature declined steadily to normal on the fifth day. It remained normal until the eighth day and then began to ascend. A culture of the pleural fluid on the tenth day showed hemolytic *Staph. albus*. It was evident that empyema was established. An open thoracotomy for drainage was done on the fourteenth postoperative day. The patient was discharged shortly thereafter and made an uneventful recovery. The pathologic report was bronchiectasis with cavitation.

The patient was sensitive to antibiotics and sulfanilamide. Streptomycin and penicillin caused such a violent bronchial spasm that neither could be used. However, the sulfanilamide placed in the pleural cavity was tolerated. From the culture reports one could reason that the mixture killed off the bacteria that originally contaminated the pleural space. A thoracotomy incision through an old thoracoplasty site is necessarily associated with a great deal of surgical trauma and a considerable amount of scar tissue that is often partially devitalized. The approximation of such tissue is not accurate when the wound is closed. Naturally, the chest wall is inoculated with the bacteria as well as the pleural cavity. The mixture placed in the pleural cavity cannot reach the bacteria in the tissues. It may be assumed that since the tissues were not aided by antibiotics, the bacteria created an infection in the chest wall tissues that spread to the pleural cavity on or about the eighth postoperative day.

The fourteen patients reported upon were given sulfanilamide by mouth for forty-eight hours before operation, and all received injections of penicillin or streptomycin, or both, after operation. Penicillin or streptomycin was not instilled into the pleural cavity.

It is thought that the mixture, when used as described will kill off all the bacteria that have been spilled into the pleural cavity at the time of operation. The mixture of azochloramide, sodium tetradecyl sulfate, and sulfanilamide is considered nonspecific in its bactericidal action. Therefore, it is much more desirable in treating a grossly contaminated pleural cavity than would be one of the antibiotics, such as penicillin or streptomycin, which is highly specific in its bactericidal action.

A rather extensive clinical trial of the mixture in treating pyogenic empyema has revealed that it will eliminate most types of pyogenic bacteria from the pleural cavity. After empyema has been established, the mixture will control but will not eliminate the hemolytic *Staph. albus* or *aureus*. It is hoped that it will eliminate these organisms if the attack against them is made before the organism actually invades the body tissues. It is reasonable to expect that contaminating bacteria would be more easily killed if attacked before they have had time to invade the body tissues.

The mixture placed in the pleural cavity can in no way influence bacteria contaminating the soft tissues of the chest wall. Therefore, it is extremely important that the patient receive the aid of penicillin and streptomycin in combating bacteria that contaminate the soft tissues of the chest wall. It is generally believed that penicillin and streptomycin will prevent infection from occurring in the soft tissue incisions. However, it should be realized that the parenteral administration of penicillin or streptomycin will reach the pleural fluid in small quantities.¹⁰

The objection to the preliminary report has not been overcome.⁴ Complete bacteriologic studies are not available. In a surgical practice carried on in several different hospitals it is difficult to avoid the loss of specimens for culture, and under the stress of surgical work cultures sometimes are not taken.

Since the culture reports are favorable it is better to disregard these reports. In each case, however, pus was seen to spill into the pleural cavity. In every instance the pleural cavity and the wounds were closed without drainage. Only one patient out of fourteen developed empyema and that patient could not tolerate penicillin or streptomycin.

In eleven of the patients reported, the contamination occurred from an inadvertent tear into an abscess cavity. The bronchial stump clamp slipped from the divided bronchus in two cases, and the bronchial secretions which were assumed to be infected were seen to contaminate the pleural cavity. The postoperative pathologic diagnoses in the thirteen patients successfully treated were: carcinoma complicated by lung abscess, four; tuberculosis, six; bronchiectasis, two; and putrid lung abscess, one.

CONCLUSIONS

Sodium tetradecyl sulfate, 1:500, in azochloramide, 1:3,300, with sulfanilamide is an efficient nonspecific bactericidal agent.

When the mixture was properly applied to the pleural cavity after pneumonectomy with gross contamination, it prevented empyema in thirteen out of fourteen consecutive cases.

REFERENCES

1. Adams, W. E., Thorton, T. E., and Carlton, L. M., Jr.: The Use of Blood Plasma for Filling the Pleural Space Following Total Pneumonectomy, *Ann. Surg.* 122: 905-916, 1945.
2. Allbritten, F. E., Jr., Flick, J. B., and Gibbon, J. H.: The Use of Sulfanilamide in Partial and Total Resection of the Lung, *J. THORACIC SURG.* 11: 187-197, 1941.
3. Blades, Brian: Penicillin as an Adjunct to the Surgical Treatment of Acute and Chronic Empyemas, *Ann. Surg.* 121: 672-677, 1945.
4. Brantigan, O. C., and Rigdon, H. L.: Sodium Tetradecyl Sulfate, Azochloramide, and Sulfanilamide Mixture in the Prevention of Empyema Following Pneumonectomy With Contamination, *J. THORACIC SURG.* 16: 416-421, 1947.
5. D'Abreu, A. L., Litchfield, J. W., and Thomson, Scott: Penicillin in the Treatment of War Wounds of the Chest, *Brit. J. Surg. (Supp.)* 32: 170-198, 1944.
6. Johnson, Julian: Total Pneumonectomy, *Surg., Gynec. & Obst.* 76: 460-468, 1943.
7. Neter, E.: Combined Bacteriostatic Activity of Sulfanilamide and Azochloramide Upon Group A Hemolytic Streptococcus and Enterococcus, *Proc. Soc. Exper. Biol. & Med.* 47: 303-305, 1941.
8. Petroff, S. A., and Schain, Philip: The Enhancement of Bactericidal Properties of Well-Known Antiseptics by Addition of Detergents, *Quart. Bull. Sea View Hosp.* 5: 372-384, 1940.
9. Strieder, J. W.: Surgical Indications and Treatment of Primary Cancer of Lung, Bronchiectasis and Lung Abscess, *M. Clin. North America* 29: 1282-1293, 1945.
10. Valle, A. R., and White, M. L., Jr.: Penicillin in Pulmonary Resection, *J. THORACIC SURG.* 19: 437-444, 1945.
11. White, W. L., Burnett, W. E., Bailey, C. P., Rosemond, G. P., Morris, C. W., Favorite, G. O., Spaulding E. H., Bondi, Amedeo, Jr., and Fowler, R. H.: Use of Penicillin in Prevention of Postoperative Empyema Following Lung Resection, *J. A. M. A.* 126: 1016-1024, 1944.

THE MECHANISM OF HEALING OF LUNG TISSUE AND ITS REACTION TO DIFFERENT SUTURE MATERIALS

ANIBAL ROBERTO VALLE, M.D.,* AND G. BURROUGHS MIDER, M.D.†
CHARLOTTESVILLE, VA.

LITTLE has been reported about the healing process of lung wounds or the reaction of pulmonary parenchyma to various suture materials. Olch and Ballou⁴ in 1929 made linear incisions in the lungs of dogs and sutured them. One-half of the dogs received intratracheal injections of trypan blue after the operation. The dye was injected to determine the source of the scar tissue formation. It was found that the dye collected in pneumonic-like patches in the lung and that practically no pigment-bearing cells were found in the scar tissue. They reported that the scar produced in the lung, the result of a simple incision and suture, resembled a scar elsewhere in the body. More recently Montgomery³ studied the healing of experimental wounds in the lung and the regenerative power of lung parenchyma. He cut a wedge, 3 cm. long on each side, out of the lung of adult cats. Bleeders, when encountered, were controlled by mattress sutures of silk, but he avoided the clamping and ligature of vessels as much as possible in order that the tissue might not be crushed. The visceral pleura was carefully approximated by interrupted silk stitches. The animals were killed at intervals of 1 to 112 days after operation. Histologic sections revealed well-formed bronchial buds at the apices of the wounds, which he thought indicated a regenerative phenomenon. He also reported the re-formation of the pleural membrane.

We have studied the reaction of the lung parenchyma of dogs to six different suture materials and their effects on healing.

METHODS

Thirty-two dogs were used in this experiment. Their average weight was eight kilograms. The sutures tried were catgut, chromic catgut, silk, linen, nylon, and cotton. The tubed nonabsorbable suture material was soaked in 95 per cent alcohol for about one hour and then autoclaved. Jenkins and Dunham¹ recommended this procedure because they found that tubing fluids often contained xylene and other highly irritating substances which cause considerable tissue reaction. The catgut was soaked in sterile water to make it pliable. The anesthesia used was intravenous nembutal in a dose of 30 mg. per kilogram of body weight. Positive pressure was maintained by an intratracheal catheter attached to a specially designed Foregger anesthesia apparatus. Sterile technique was used throughout.

In this set of experiments we carried out a technique identical with that used in pulmonary resection in man. The dog's chest was shaved and prepared with tincture of merthiolate. An incision approximately 10 cm. long was made along the fourth rib. The fourth rib was removed subperiosteally, and the pleural cavity opened. A lobe of the lung was brought to the outside. One or sometimes two Kelly clamps were placed near the periphery of the lobe and a slice about 2 cm. wide was removed distal to the clamp. A

From the Departments of Surgery and Gynecology, and Pathology, University of Virginia School of Medicine, and University Hospital.

Received for publication Oct. 4, 1948.

*Now at Fitzsimons General Hospital, United States Army, Denver, Colo.

†Now at University of Rochester School of Medicine and Dentistry, Rochester, N. Y.

suture was tied at one end and a continuous stitch placed around the clamp or clamps. The clamp was removed and each stitch tightened individually before the other end was secured. A few bleeders were encountered, which were clamped and tied with the same suture material. The lung was then inflated with a pressure equal to 15 cm. of water, and the chest was closed in layers without drainage. Silk was used throughout in the chest closure. No dressing was applied.

Two operations were performed on most of the dogs, the second suture being placed in a different lobe. When the specimen was taken, the whole lobe was removed. The specimens were taken at intervals of 24, 48, and 72 hours, and 7, 14, 30 and 60 days.

We performed a second set of experiments in which no lung tissue was resected but the suture material was simply passed through the lung parenchyma. The suture material was threaded through a long needle, knotted at one end, and then knotted at the other end after being passed through the lung. The same studies were made on these specimens which were taken at 3, 7, and 14 days.

Several representative blocks of tissue were cut from each specimen. They were fixed in Zenker's fluid and imbedded in paraffin. Histologic sections were stained with hematoxylin and eosin, Masson's trichrome stain (after Goldner's modification), and Weigert's stain for elastic tissue.

WOUND HEALING

Repair in the pulmonary wounds proceeded in a characteristic manner.

Twenty-four Hours.—The pleural surface of the wound was covered by a thin layer of fibrin which extended over the hyperemic pleura for several millimeters on either side of the suture line. The cut edges of the pleura could be traced into the wound which appeared as a mass of hemorrhagic, necrotic tissue containing few recognizable alveoli and bronchioles. The pulmonary alveoli of the adjacent hyperemic lung were filled with blood. Small numbers of polymorphonuclear leucocytes were seen at the junction of the necrotic tissue and the blood-filled pulmonary alveoli. Small arteries and veins contained fresh thrombi, and some of the bronchioles and smaller bronchi contained a mixture of blood and fibrin in which only a few leucocytes were seen. There was no evidence of fibroblastic proliferation in any of the sections studied.

Forty-eight Hours.—The wound contained more polymorphonuclear leucocytes but was still bloody. The fibrous stroma in the adjacent normal lung appeared edematous. Bronchioles within the wound area and in the contiguous pulmonary tissue were lined by flattened epithelial cells. Some smaller bronchi were lined by two or three layers of polygonal cells which resembled closely those in the stratum germinativum of epidermis although no intercellular bridges could be seen. Mitotic figures were numerous among the cells lining the affected bronchi and bronchioles.

Seventy-two Hours.—Blood was less conspicuous in and about the wound. There was marked proliferation of all the fibroblasts in the stroma of the region. The alveolar walls of the adjacent lung were thickened. Apparently the peribronchial, perivascular, subpleural, and interlobular connective tissues all contributed to this response which extended for several millimeters into the normal lung. There was no leucocytic reaction in the proliferating areas and the leucocytes within the hemorrhagic and necrotic center of the wound were still polymorphonuclear. The epithelial cells lining the affected bronchi and bronchioles appeared to be squamous in type, but the lumina of these structures were well preserved. Fibroblasts had invaded the intrabronchial exudate, and organization was apparently proceeding without the presence of leucocytes.

Seven Days.—The core of the wound was now a mass of hemorrhagic exudate containing polymorphonuclear leucocytes, surrounded by a broad zone of vascular granulation tissue. There were moderate numbers of lymphocytes and plasma cells in some areas. No vestige of the damaged pulmonary alveoli remained, but there were focal accumulations of squamous epithelial cells surrounded by thin capsules which represented metaplastic bronchioles, and occasionally a typical bronchus was found lined by stratified squamous epithelium in the region of the wound (Fig. 1). There was much less blood in the wound

than formerly. The pleural splint was being organized by fibroblasts from the subpleural connective tissue, but the cut edges of the pleura could still be traced into the substance of the wound and their elastic fibers did not appear to be altered. A collagen-like substance was present in the thickened walls of the alveoli lying in the zone of fibroblastic proliferation seen at the end of 72 hours. Small perivascular accumulations of lymphocytes, plasma cells, and macrophages were seen in the lung tissue extending beyond the zone in which the alveolar walls were altered.

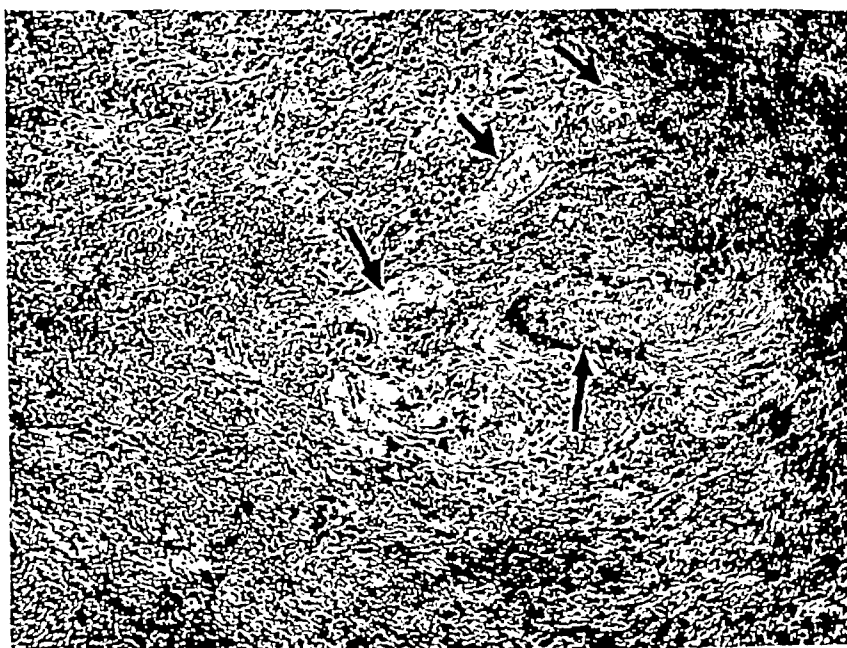


Fig. 1.—Squamous metaplasia of bronchiolar epithelium in healing wound of dog's lung 10 days after operation. (Hematoxylin and eosin stain, $\times 100$.)



Fig. 2.—Healing wound of dog's lung 10 days after operation. Granulation tissue unites the cut edges of pulmonary tissue and an organizing fibrinous exudate still splints the pleural surface. (Hematoxylin and eosin stain, $\times 60$.)

Fourteen Days.—Most of the wound area was a mass of young vascular scar tissue containing a few macrophages, lymphocytes, and plasma cells. Occasional bronchi were seen imbedded in the wound area. Most of them were lined by columnar epithelium, only a few by squamous cells. Some collapsed medium-sized blood vessels could be made out in the young scar tissue. The cut edges of the pleura could still be traced into the wound in sections stained to show elastica. The pleural splint was almost entirely organized (Fig. 2). A few small circumscribed foci of squamous epithelial cells and, occasionally, typical dilated bronchi lined by columnar cells were seen in the newly formed scar.

Thirty Days.—The wound area was considerably smaller and contained much collagen. The scar tissue was less vascular than at 14 days. Hyalinized remnants of bronchi and blood vessels and a few thin-walled bronchi filled with mucus and lined by columnar epithelium were imbedded in the scar tissue in several of the wounds studied (Fig. 3). The



Fig. 3.—Healed wound of dog's lung 30 days after operation. Adult-type scar tissue has effected closure of the wound. The scar contains a trapped bronchiole filled with secretion and a thrombosed and recanalized artery. (Hematoxylin and eosin stain, $\times 60$.)

elastica of the cut edges of the pleura was still visible. There was no evidence of regeneration of the pleura or its elastica in the depths of the wound. The pleural surface was covered by a layer of mesothelial cells. In one wound there were hemosiderin-laden macrophages about the scar, but generally hemosiderin deposits were not seen at any stage in the healing process. Strassmann has shown that blood injected into rabbit's lung may disappear rapidly without the local formation of hemosiderin.

INFLUENCE OF SUTURE MATERIAL ON HEALING

It should be remembered that the suture in each of these wounds had a dual function, apposition of cut surfaces and hemostasis. Each suture lay in fibrous tissue, usually peribronchial stroma. A zone of stroma and parenchyma 1 to 2 mm. wide between the suture and the wound was compressed as the result of the tension necessary for hemostasis (Fig. 4). This local reaction may have modified the healing process, but the assumption that any such effect of tension was relatively constant for all of the wounds studied seems to be warranted.

Wounds sutured with nylon appeared consistently larger and contained more histologic evidence of inflammatory reaction at all stages of development studied than did wounds closed with the other materials used. The nylon wounds still protruded above the pleural surfaces at one month when other wounds were depressed. The zone of hyperemia persisted

longer and was always wider. Considerably more scar tissue was formed, but there was no evidence that the process of collagen deposition was delayed.

The wounds closed with cotton and linen sutures contained more leucocytes than those in which silk or catgut was used. The inflammatory cells were still present in appreciable numbers at 30 days when cellular inflammation had almost completely disappeared from the other wounds. No incontrovertible evidence of delayed scar formation was found, however.

No essential differences could be seen in the histologic appearances of the wounds sutured with silk, plain catgut, or chromic catgut, save for a transitory rise in inflammatory reaction in the catgut wounds during the first week of the healing process. Absorption



Fig. 4.—Sutured wound of dog's lung 2 days after operation. Note general resemblance to infarct. (Hematoxylin and eosin stain.)

of the catgut led to isolated foci of foreign body and chronic inflammatory reaction about the suture area, but this process produced no notable change in the wounds.

REACTION ABOUT THE SUTURES IN THE WOUNDS

The tension on the sutures may have been responsible for some of the differences observed in the degree of inflammatory reaction about the suture material used to close the pulmonary wounds. The various suture materials were associated with the following reactions (Fig. 5):

Silk.—A few polymorphonuclear leucocytes appeared during the latter part of the first week, but most of them had disappeared at the end of the second week when there was some proliferation of the surrounding tissue. At the end of 30 days the suture was imbedded in a thin capsule of fibrous tissue, and no leucocytes were seen in the region.

Plain Catgut.—Polymorphonuclear leucocytes appeared about the suture during the third day and were moderately numerous at the end of a week. The sutures had been almost completely absorbed by the end of two weeks when the remnants were surrounded by foreign body giant cells, a few round cells, and a thin zone of fibrous tissue. One month after the wound was made, small fibrous nodules containing a few foreign body giant cells indicated where the catgut had been previously.

Chromic Catgut.—Small numbers of polymorphonuclear leucocytes were seen about the suture until the end of the second week when the catgut had become fragmented and was surrounded by foreign body giant cells, round cells, and proliferating fibroblasts. The

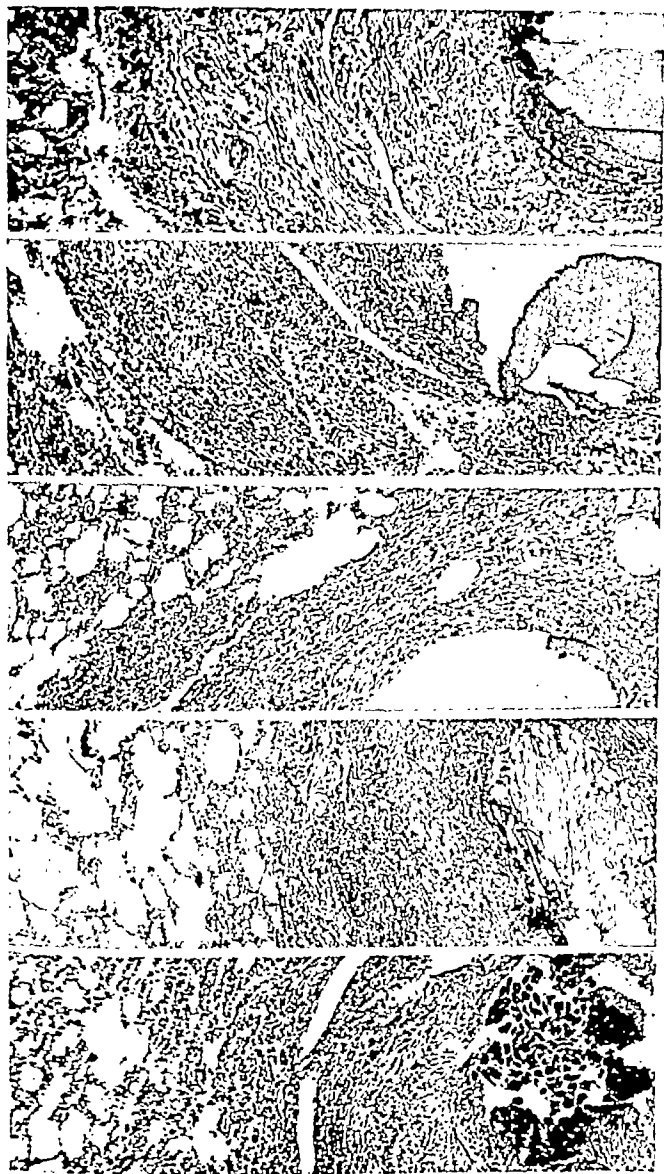


Fig. 5.—Tissue reaction about suture material used to close wounds of dog's lung 11 days previously. *A*, Silk; *B*, cotton; *C*, nylon; *D*, chromic catgut; *E*, linen.

fragments were smaller in specimens examined at 30 days, but the inflammatory reaction was similar.

Linen.—Many polymorphonuclear leucocytes were found in and about the suture material 24 hours after the wound was made. These cells persisted forming a dense collection about the linen so that the suture appeared to be in the center of a tiny abscess at the end of the first week. Thereafter the abscess-like lesion persisted and was walled off by fibrous tissue at the end of 30 days.

Cotton.—The changes were almost precisely the same as those described for linen.

Nylon.—At the end of 24 hours the nylon sutures were surrounded by a zone of necrosis as much as 100 microns wide. The degree of cellular inflammatory reaction found subsequently varied greatly. The constant change was a marked proliferation of fibrous tissue which enclosed the nylon in a fibrous nodule four to five times as large as the encapsulated silk suture at 30 days.

REACTION TO SUTURE MATERIAL ALONE

The various suture materials were imbedded in normal lung tissue by passing them approximately 2 cm. below the pleura and tying the ends loosely together over the pleural surface. In this way the reaction of the pulmonary components to the foreign bodies could be studied without the added effects that might have been due to closure of the wounds. Particular attention was paid to the nature and quantity of inflammatory exudate, fibroblastic proliferation, and deposition of collagen.

The changes observed about the silk, catgut, linen, cotton, and nylon were similar in all details to those described about the sutures in the healing wounds.

COMMENT

The technique employed in producing the wounds we studied produced a hemorrhagic necrotic mass which was organized by fibroblastic proliferation. The general principles seem to be the same as those described for wounds healing by second intention in other parts of the body. The structural changes observed have been described previously by Montgomery,³ whose investigations on the healing of lung wounds are the most complete we have found. His procedure on cats was less drastic than the one used in these studies. Perhaps this may account for failure of the pleura to regenerate its elastic tissue in our dogs.

These studies lend no support to the thesis that pulmonary tissue regenerates itself in the process of repairing a parenchymal wound. Montgomery described the pneumatization of scar tissue by proliferation of bronchial buds and the formation of new alveoli. He assumed that spaces lined by cuboidal epithelial cells were newly formed bronchial buds. Cells resembling cuboidal epithelium line the thick-walled alveoli adjacent to the wound area after one week. The thickening of the walls is due to the appearance of a substance which has the staining characteristics of collagen but is not fibrillar. While the week-old wound contains many thick-walled alveoli lined by cuboidal cells, these become less numerous in the wounds studied at two weeks and one month. Only a few persist at two months. This suggests that the change in type of lining epithelium may be caused by an alteration in the environment of the lining cells occasioned by alteration in their supporting tissue. Apparently the collagen-like substance disappears as healing progresses, and most of the alveoli are reconstructed in their original form.

Montgomery stated that buds from the bronchi aerated the scar, indicating regeneration of pulmonary parenchyma. Our studies indicate that the chief mechanism by which the scar becomes smaller is through the contracture of scar tissue. Recognizable bronchioles and bronchi were more numerous within the wound during the first two weeks after operation than in the later stages of healing. Recognizable alveoli were seldom found in the scar. The reduction of the numbers of parenchymal units in the scar tissue militates against the thesis that aeration is accomplished by invasion of bronchial buds. It seems more probable that parenchymal remnants are trapped in the wound and become imbedded in scar tissue.

The suture material used had little influence on the rate of healing of the experimental pulmonary wounds in dogs. The end result was similar in all cases. Certain differences in the degree of inflammatory reaction and fibroblastic proliferation may be significant on theoretical grounds. If the operative field were not aseptic it is possible that the increased intensity of cellular inflammatory reaction due to the sum of the effects of the infecting agent and the suture material might be more conducive to necrosis. It is probable, however, that the necrotizing potentialities of the infecting organism are more important than the inflammatory component elicited by the suture material. Nylon, however, seems to us to be a poor substance to introduce into the lung. All of the wounds closed with nylon were much larger at all stages of healing than were those sutured with other materials. Therefore, these wounds might provide a better medium for the growth of bacteria. The excessive amount of scar tissue formed in the nylon wounds seems undesirable. Scar tissue in large quantities is essentially weak. This may be of little practical significance in the lung where physiologic stresses are usually of a low order of magnitude. It would seem unwise to use nylon when other readily available suture materials produce none of these theoretically objectionable changes.

The results of this work seem to indicate that the most desirable suture materials for lung tissue are silk, plain catgut, and chromic catgut.

Acknowledgment.—We wish to express our appreciation to Johnson & Johnson, New Brunswick, N. J., who furnished the Ethicon sutures used in this work.

REFERENCES

1. Jenkins, H. P., and Dunham, C. L.: Irritant Properties of Tubing Fluids as a Factor in the Tissue Reactions Observed With Surgical Gut (Catgut), *Ann. Surg.* 118: 288, 1943.
2. Langston, H. T.: The Problem of Catgut Sensitivity and Its Relation to Wound Healing, *Ann. Surg.* 115: 141, 1942.
3. Montgomery, J. L.: Healing of Experimental Wounds of Lung, *Brit. J. Surg.* 31: 292-299, 1944.
4. Olch, I. Y., and Ballou, H. C.: The Origin of Scar Tissue in Healing of the Lung, *Arch. Surg.* 19: 1595, 1929.
5. Strassman, G.: Formation of Hemosiderin in the Lungs, *Arch. Path.* 38: 76, 1944.

NOTICES

TUMOR REGISTRY COMMITTEE

The Tumor Registry Committee was unable to obtain the necessary assistance for the program it was instructed to carry out. Unwilling to appear empty-handed at the Denver meeting, the chairman will attempt to gather data for the more limited objective which he suggested at the New Orleans meeting: The relationship of interval and late results of operation to the cancerous lesion. Dr. Neuhoof and Dr. Aufses, aided by a Research Fund grant from the Mount Sinai Hospital, propose to make an inquiry among all the members of the Association in a form which will be simple and not time-consuming. They anticipate sufficient material for a report at the April meeting at which time the Association may decide if further development of the method would be a useful function of the Tumor Registry. All members are urged to respond even if there are only single cases to report.

THE JAMES ALEXANDER MILLER FELLOWSHIP FOR RESEARCH IN TUBERCULOSIS

The New York Tuberculosis and Health Association announces that a fellowship will be available from July 1, 1950, to June 30, 1951. The fellowship is designed to support a qualified medical investigator who will devote full time to a research project with a definite bearing on tuberculosis. Assurance must be provided that the applicant will be acceptable in the laboratory or clinic of his choice and that he will be provided with the facilities necessary for the pursuit of the work. It is preferred that such laboratory or clinic be located in New York, N. Y.

The stipend will be \$5,000.

Application forms will be supplied on request to the New York Tuberculosis and Health Association, 386 Fourth Avenue, New York 16, N. Y., and must be submitted in duplicate not later than March 1, 1950. Announcement of the award will be made in April, 1950.

BOARD OF THORACIC SURGERY

In June, 1949, the Secretary of the Board of Thoracic Surgery met with the American Board of Surgery, and the problem of those individuals who are neither members of The American Association for Thoracic Surgery nor diplomates of the American Board of Surgery was considered. The American Board of Surgery allowed a new group to be admitted to the Founders Group at the discretion of the Board of Thoracic Surgery. This group will be known as group "D" and will consist of other surgeons who are not members of The American Association for Thoracic Surgery or diplomates of the American Board of Surgery and whose training, because of the period in which it was taken, does not qualify them to become certified by the American Board of Surgery, but who may upon careful scrutiny and adequate evidence of proficiency be eligible for consideration as founder members.

ASSOCIATION MEETING TRANSPORTATION

The Burlington Railroad has offered to set aside some pullman space on the Denver Zephyr leaving Chicago 5 P.M., April 13, 1950, for our membership enroute to Denver. This space will be held by Mr. William Moloney, General Agent, Passenger Department, 105 W. Adams Street, 711-712 Bankers Building, Chicago 3, Ill. (phone Wabash 2-2345). Reservations may also be obtained from your nearest Burlington representative in principal cities throughout the United States. At the same time, if desired, return reservations may be made. The Burlington representative, Mr. A. G. Matthews, will be on hand at the meeting Friday, January 13, in Dr. Harper's office in the Republic Building.

The Journal of Thoracic Surgery

VOL. 19

MARCH, 1950

No. 3

Original Communications

MEDIASTINAL "TUBERCULOMA"

SURGICAL REMOVAL IN FOUR PATIENTS

PAUL C. SAMSON, M.D.,* OAKLAND, CALIF., BRIGADIER GENERAL LEONARD D.

HEATON, UNITED STATES ARMY† (BY INVITATION), AND DAVID J.

DUGAN, M.D.,‡ OAKLAND, CALIF.

WITH the passage of years, detailed reviews of pathology and treatment have increased our common knowledge of mediastinal tumors. Mediastinal granulomas have received little attention, however, which undoubtedly bespeaks their rarity.

In earlier reports on mediastinal tumors, the differential diagnosis from mediastinal masses due to tuberculosis or syphilis is mentioned. The impression is gained that the distinction should not have been difficult since concomitant signs of specific disease would be present in addition to the central chest lesion.^{1, 2} Apparently, Piazzzi-Martinni³ was the only author actually to describe cases of mediastinal tuberculoma and gumma. Heuer⁴ has excluded both tuberculous and nontuberculous mediastinal lymphadenitis from the consideration of mediastinal tumors. It was not stated how the former diagnoses were made. More recently, Grow and co-workers⁵ discussed eight instances of "tuberculous lymphadenopathy" among seventy-three patients who were explored because of mediastinal lesions. No further details were given.

Only two descriptions of the surgical extirpation of mediastinal tuberculomas have been found. Grace's case⁶ apparently was the first to be recorded. The pathologic diagnosis was unsuspected prior to removal. The patient had a slightly positive tuberculin reaction. No special search was made for tubercle bacilli in the specimen. The patient was well at last report. Blades and Dugan⁷

*From the Surgical Services of Letterman General Hospital and Lane-Stanford Hospital, San Francisco, Calif.

Read at the Twenty-ninth Annual Meeting of The American Association for Thoracic Surgery, New Orleans, La., March 29-31, 1949.

*Civilian Consultant in Thoracic Surgery, Letterman General Hospital; Associate Clinical Professor of Surgery, Stanford University School of Medicine.

†Chief of Surgical Service; Chief of Professional Services, Letterman General Hospital, San Francisco, Calif.

‡Consultant in Thoracic Surgery, Veterans Administration Hospital, Oakland, Calif.

NOTICES

TUMOR REGISTRY COMMITTEE

The Tumor Registry Committee was unable to obtain the necessary assistance for the program it was instructed to carry out. Unwilling to appear empty-handed at the Denver meeting, the chairman will attempt to gather data for the more limited objective which he suggested at the New Orleans meeting: The relationship of interval and late results of operation to the cancerous lesion. Dr. Neuhoef and Dr. Aufses, aided by a Research Fund grant from the Mount Sinai Hospital, propose to make an inquiry among all the members of the Association in a form which will be simple and not time-consuming. They anticipate sufficient material for a report at the April meeting at which time the Association may decide if further development of the method would be a useful function of the Tumor Registry. All members are urged to respond even if there are only single cases to report.

THE JAMES ALEXANDER MILLER FELLOWSHIP FOR RESEARCH IN TUBERCULOSIS

The New York Tuberculosis and Health Association announces that a fellowship will be available from July 1, 1950, to June 30, 1951. The fellowship is designed to support a qualified medical investigator who will devote full time to a research project with a definite bearing on tuberculosis. Assurance must be provided that the applicant will be acceptable in the laboratory or clinic of his choice and that he will be provided with the facilities necessary for the pursuit of the work. It is preferred that such laboratory or clinic be located in New York, N. Y.

The stipend will be \$5,000.

Application forms will be supplied on request to the New York Tuberculosis and Health Association, 386 Fourth Avenue, New York 16, N. Y., and must be submitted in duplicate not later than March 1, 1950. Announcement of the award will be made in April, 1950.

BOARD OF THORACIC SURGERY

In June, 1949, the Secretary of the Board of Thoracic Surgery met with the American Board of Surgery, and the problem of those individuals who are neither members of The American Association for Thoracic Surgery nor diplomates of the American Board of Surgery was considered. The American Board of Surgery allowed a new group to be admitted to the Founders Group at the discretion of the Board of Thoracic Surgery. This group will be known as group "D" and will consist of other surgeons who are not members of The American Association for Thoracic Surgery or diplomates of the American Board of Surgery and whose training, because of the period in which it was taken, does not qualify them to become certified by the American Board of Surgery, but who may upon careful scrutiny and adequate evidence of proficiency be eligible for consideration as founder members.

ASSOCIATION MEETING TRANSPORTATION

The Burlington Railroad has offered to set aside some pullman space on the Denver Zephyr leaving Chicago 5 p.m., April 13, 1950, for our membership enroute to Denver. This space will be held by Mr. William Moloney, General Agent, Passenger Department, 105 W. Adams Street, 711-712 Bankers Building, Chicago 3, Ill. (phone Wabash 2-2345). Reservations may also be obtained from your nearest Burlington representative in principal cities throughout the United States. At the same time, if desired, return reservations may be made. The Burlington representative, Mr. A. G. Matthews, will be on hand at the meeting Friday, January 13, in Dr. Harper's office in the Republic Building.

Right posterior thoracotomy was performed on May 19, 1947, through the fourth intercostal space. The upper lobe was adherent to a globular mass which measured 3.5 by 3 cm. The tumor was cystic and the wall was thick and yellowish in color. It was situated lateral to the trachea and was adherent to this structure and to the posterior aspect of the innominate artery. The vagus nerve coursed deep within the capsule and was sacrificed (Fig. 2). Medially and inferiorly the mass was bound down to several calcified lymph nodes which extended in a chain anterior to the trachea and toward the left. Due to extreme adherence, the nodes to the left of the trachea were not removed.

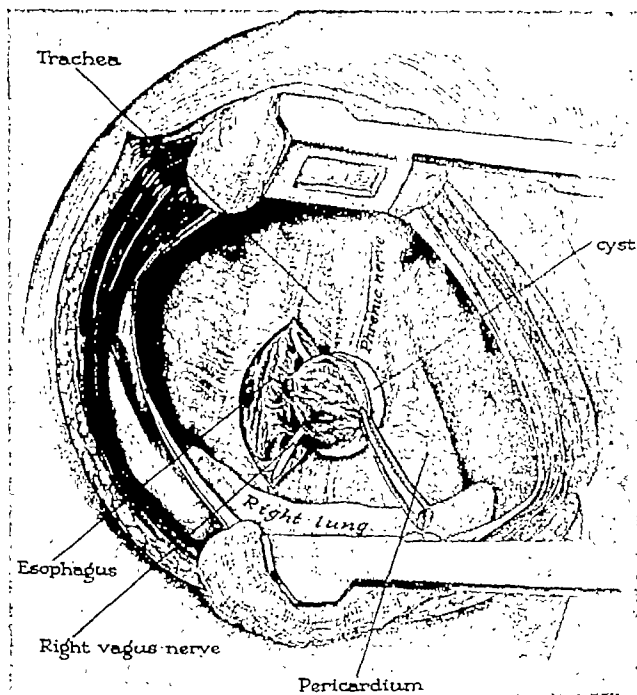


Fig. 2 (Case 1).—Drawing at operation showing position of mass in relation to mediastinal structures.

The postoperative recovery was uncomplicated and the patient left the hospital twelve days later.

Grossly, the cyst contained white caseous material. Numerous flecks of calcium were encountered in the contents and also in the thick fibrous wall. Microscopic examination showed fibrosis and chronic inflammation (Fig. 3). No epithelial lining could be seen. Culture of the contents of the cyst was negative for acid-fast organisms.

CASE 2.—J. L., a white man, aged 19 years, had no symptoms referable to his chest. On roentgen examination prior to separation from the Army, a left-sided, oval, anterior mediastinal mass was discovered (Fig. 4).

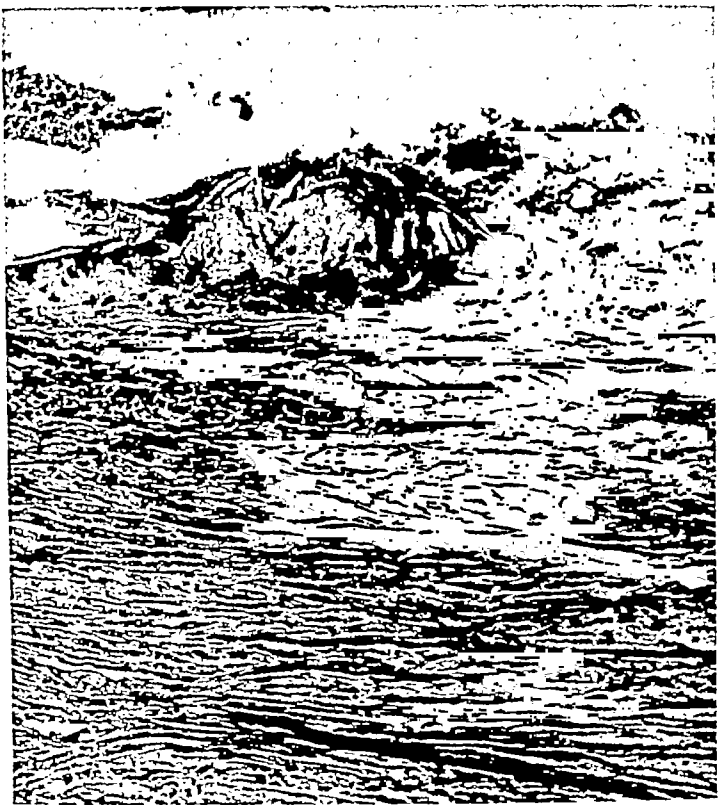


Fig. 3 (Case 1).—Photomicrograph $\times 100$. Fibrous capsule showing cellular infiltrate and calcific deposits surrounding zone of caseation.



Fig. 4 (Case 2).—Roentgenogram, posteroanterior and left lateral projections. An oval mass measuring 2.5 by 5 by 5 cm. is present in the left anterior mediastinum. There are small scattered areas of increased density suggestive of calcification.

Except for moderate obesity, the physical examination was unrevealing. There was no lymphadenopathy. The blood and urine were normal. The basal metabolism rate was -14. Both first and second strength PPD tuberculin tests were negative prior to and following operation. The coccidioidin skin test, 1 to 1000, was negative. The scanty mucoid sputum was inoculated into a guinea pig and no acid-fast bacilli were found.

Serial roentgenograms showed no change in the size of the tumor. Suggestive calcified densities were seen in some of the films and three possible diagnoses were entertained: dermoid cyst, tuberculous lymph nodes, and tumor of the lymphoma group.

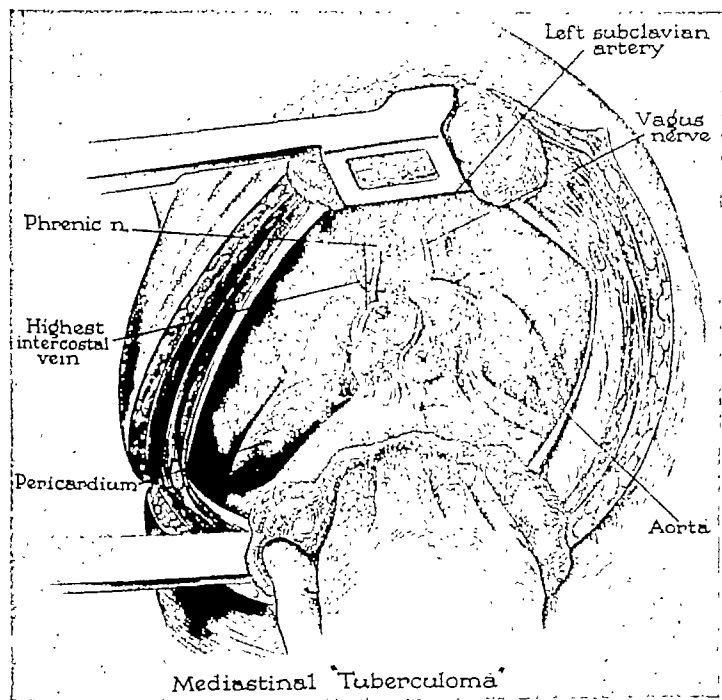


Fig. 5 (Case 2).—Drawing at operation showing lobulated and cystic mass in the left hilar region.

Left posterior thoracotomy was performed on June 13, 1947. There were no adhesions. The upper lobe was normal except for a small irregular subpleural calcified area in the anterior segment. The encapsulated mediastinal mass lay at the hilum beneath the mediastinal pleura and within the curve of the aortic arch. It measured 8 by 5 cm., was lobulated and partially cystic (Fig. 5). The vagus nerve was carefully dissected free from the posterior capsule. The phrenic nerve which coursed over the anterior surface of the mass was stretched somewhat during separation. Inferiorly and medially the mass was adherent to the pulmonary artery.

The postoperative course was uneventful. On discharge, the chest was normal except for paresis of the left diaphragm. The patient has remained well.

Grossly, the mass was encapsulated and nodular, suggesting the fusion of at least three lymph nodes. Creamy white fluid and caseous material were contained in the cystic nodules. Small plaques of calcium were present in the wall. Microscopically, there was extensive caseation necrosis with an occasional rim of lymphoid tissue remaining at the periphery. Some of the caseous foci were enclosed only by a fibrous wall which was also undergoing caseation necrosis. Scattered calcifications were present in both caseous and granulomatous areas. Some caseous foci were enclosed by a zone of epithelioid cells through which were scattered many multinucleated giant cells varying from Langhan's to the foreign body type. In one area, small epithelioid tubercles were identified in the remaining lymphoid tissue (Fig. 6).

Special stains revealed no acid-fast organisms and cultures of material from the mass were negative for tubercle bacilli.



Fig. 6 (Case 2).—Photomicrograph $\times 100$. A rim of lymphoid tissue is present. A zone of epithelioid cells with one multinucleated giant cell borders the area of caseation necrosis seen at the bottom.

CASE 3.—Private DiL., a white man, aged 22 years, was admitted to Letterman General Hospital on Oct. 1, 1946. He complained of an enlarging mass in the neck which had been present for one month. The patient had slight dyspnea when supine. Physical examination showed a soft, irregular, tender mass 5 by 2 cm. lying at the lower anterior edge of the left sternocleidomastoid muscle. The trachea was displaced slightly to the right. The mass moved but little on swallowing.

Exploration was done on the neck on October 23. The mass had penetrated the ribbon muscles and thyroid isthmus and was adherent to the trachea. It was dissected free and a connecting sinus was followed deep to the sternocleidomastoid muscle for a distance of 5 cm. This appeared to end blindly. The entire cavity was curetted, powdered with iodoform, and the skin and platysma closed. Examination of the specimen revealed a central abscess. Microscopically, the wall of the abscess showed epithelioid cells and

infiltration with polymorphonuclear leucocytes and eosinophiles. Rare giant cells of the foreign body type were present. Gram stains and acid-fast stains were nonrevealing. The guinea pig inoculation was negative for tuberculosis. The final pathologic report was encapsulated, nonspecific, chronic granuloma.

Continued swelling necessitated repeated aspirations. Three weeks later surgical drainage was performed, followed by daily irrigations. Three months following the original operation one irrigation caused severe cough. Lipiodol was then instilled in the sinus. There was continuity with a mass at the hilum of the left lung and a bronchial connection was demonstrated (Fig. 7). A secondarily infected bronchogenic cyst was suggested. Earlier thoracic roentgenograms were then reviewed which had been previously reported as normal. The first of these had been taken elsewhere two months prior to development of the suprclavicular swelling. Each showed the presence of an ill-defined mass in the left hilum just below the aortic knob. The mass was enlarging slightly. Bronchograms produced six months after the original operation showed persistence of a Lipiodol puddle in the mass but no further bronchial connection.

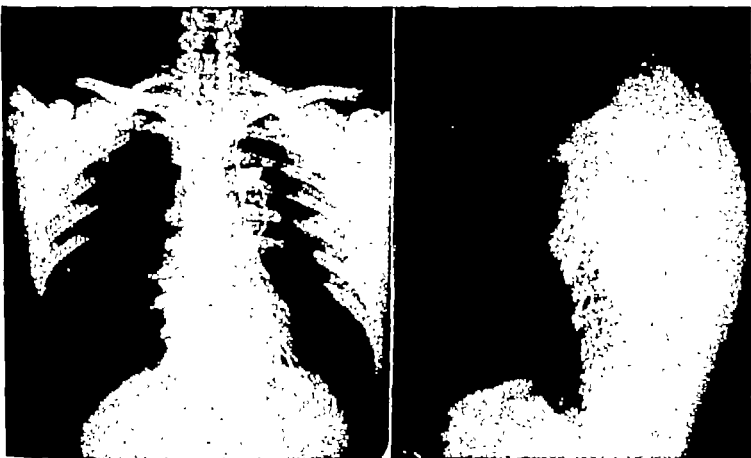


Fig. 7 (Case 3).—Roentgenogram in posteroanterior and left lateral projection following injection of Lipiodol into neck sinus. A narrow intrathoracic track is outlined leading to the mass in the left hilum where the Lipiodol is puddled. A generous bronchial communication is demonstrated.

The sinus in the neck eventually healed. Over a period of six months the patient's cough had increased appreciably.

On July 14, 1947, left-sided posterior thoracotomy was performed through the bed of the fifth rib. There were no adhesions. A 4 cm. nodular mass, partially hard and partially cystic, was found lying on the pulmonary artery incompletely covered by lung tissue. A firm cord was present beside the vagus nerve extending into the neck (Fig. 8). The vagus nerve and its recurrent branch were partially encased in the fibrous capsule of the mass. Both were dissected free with some difficulty. The phrenic nerve was inadvertently severed during separation. Removal of the entire mass was accomplished. Firm adherence resulted in a 7 mm. tear of the pulmonary arterial wall but this was repaired without difficulty. The tubular extension into the upper anterior mediastinum broke free in the region of the thyroid gland (Fig. 9). No lumen could be demonstrated nor could any connection with the bronchial tree be established.

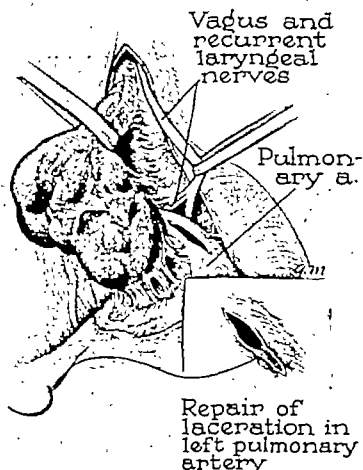
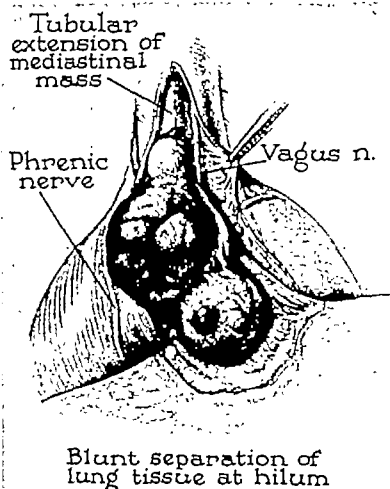


Fig. 9 (Case 3).—Details of dissection and of repair of lacerated pulmonary artery.



Fig. 10 (Case 3).—Photomicrograph $\times 50$. Small tubercle undergoing central caseation necrosis in remnant of lymphoid tissue.

Physical examination in October showed Argyll-Robertson pupils. There were no pathologic reflexes. A few clicking râles were heard over the trachea and the right upper lobe. There was a soft blowing murmur over the right second interspace and the second beat at the apex was reduplicated. The blood pressure was 120/80 in both arms. The pulse rate varied from 100 to 110 and the rhythm was regular.

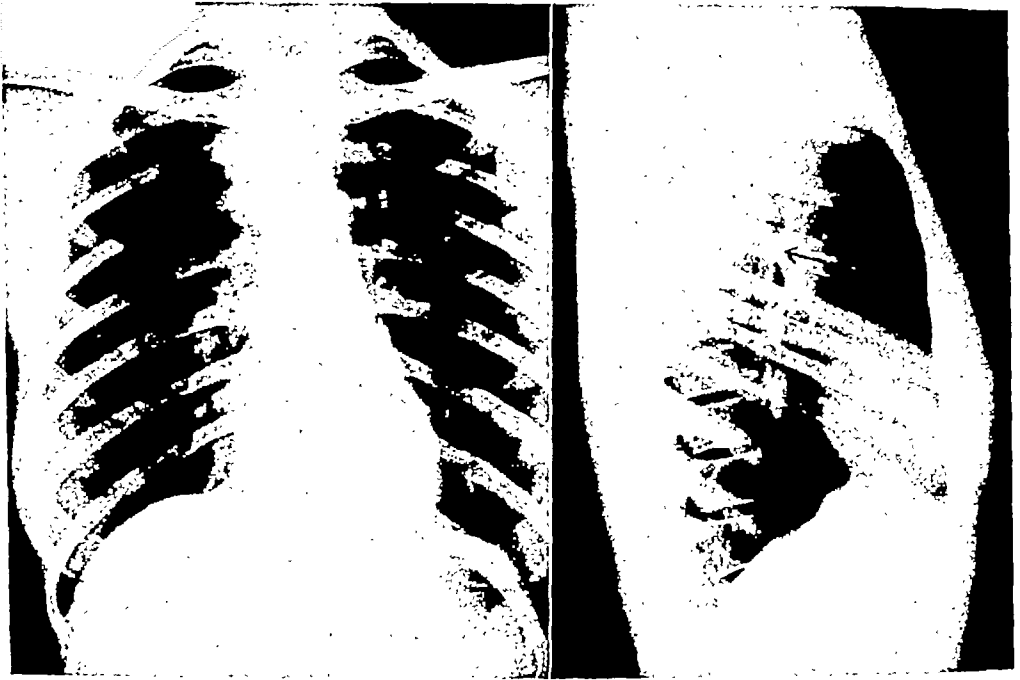


Fig. 11 (Case 4).—Roentgenogram, posteroanterior and right lateral projections. A smooth slightly lobulated mass is present in the right upper mediastinum. This has enlarged appreciably in three months time. In the lateral projection, the mass is immediately anterior to the trachea and there is suggestive compression of its lower portion.



Fig. 12 (Case 4).—Bronchoscopic view. The carina is pleated by external pressure. The tracheal lumen is reduced.

Laboratory studies revealed the following: hemoglobin, 15.5 Gm.; red blood cells, 3,920,000; white blood cells, 9,150; sedimentation rate, 52 mm. in one hour; urine, normal. The intradermal skin test to tuberculin, 1 to 1000, was negative; to coccidioidin, 1 to 1000, was negative, and to histoplasmin, 1 to 1000, strongly positive. The scanty sputum was re-

peatedly negative for acid-fast bacilli on concentrate examination. A roentgenogram of the chest showed a small, slightly rounded shadow in the mediastinum at the level of the right first interspace anteriorly. Fluoroscopically it did not seem to pulsate and there was fairly satisfactory separation from the aortic arch. Laminagrams showed no encroachment on the lumen of the trachea. The patient refused further advice at this time.

During the ensuing four months, many of the patient's symptoms became aggravated. In particular, the cough became more constant, brassy, and only partially controlled by codeine. Posttussive dyspnea and wheezing developed, and faint oral stridor could be heard. Roentgenographically, the mediastinal mass had increased in size (Fig. 11). Bronchoscopy (P.C.S.) revealed extensive anterolateral pressure on the trachea and orifice of the right stem bronchus, the lumens being reduced approximately 30 and 50 per cent, respectively. The carina was accordion pleated (Fig. 12). The walls were smooth and the bronchoscope could be maneuvered past the obstruction, which pulsed slightly.

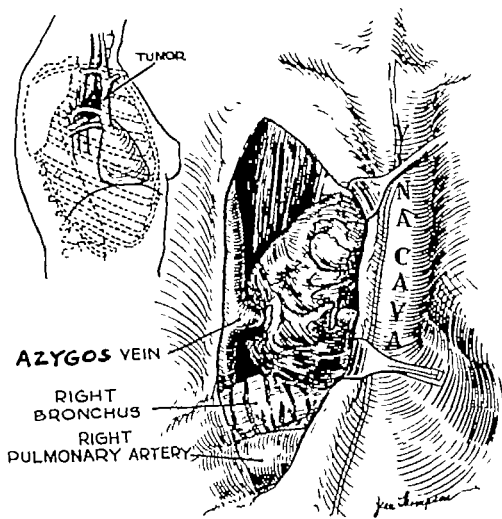


Fig. 12 (Case 4).—Drawing at operation. The mass is encroaching on the right lateral and anterior aspect of the lower trachea. The azygos vein has been divided for better exposure.

Exploration was recommended. The preoperative diagnosis was mediastinal tumor or, possibly, aneurysm.

Right anterior thoracotomy was performed* on March 3, 1948. A firm nodular mass 5 by 3 by 2 cm. was found (Fig. 13). The azygos vein was ligated to give better exposure. A portion of the mass was cystic and caseous material was extruded. Grossly, the mass was at least partially composed of matted lymph nodes. The portion which extended across the mediastinum to the left was not removed. A small tear in the superior vena cava was easily repaired. The pleural fluid removed on two occasions following operation was sterile and negative for tubercle bacilli on guinea pig inoculation.

Histologically, the tissue showed granulomas with central caseation. At the border of the necrotic tissue were numerous epithelioid cells, lymphocytes, plasma cells, and a few giant

*By Dr. Emil Holman.

cells. The granulomas were surrounded by hyalinized fibrous tissue with focal collections of lymphocytes. Small satellite tubercles were occasionally found. Pyogenic cultures were negative as were special stains to reveal acid-fast bacilli, spirochetes, and bacteria.

Eight months following operation the patient had practically no cough. Her appetite was poor and she continued to be nervous. The heart rate averaged 80 per minute. The thoracic roentgenogram was satisfactory. The intradermal tuberculin test, 1 to 100, was 2 plus.

PREOPERATIVE DIAGNOSIS AND INDICATIONS FOR EXCISION

Fairly complete information is now available on the eight known cases which have been subjected to surgery (Table I). Is it possible to make a more accurate diagnosis of mediastinal granuloma prior to operation? Survey of the available material suggests that it would be difficult. Concomitant evidence of active tuberculosis or syphilis might suggest the etiology of the mediastinal "tumor."² Roentgenograms certainly have not demonstrated "typical" masses; indeed, the shadows have uniformly mimicked more commonly encountered lesions. Evidence of scattered calcium deposits can be found in teratoid tumors as well as in granulomas. Undoubtedly, skin testing should be done more frequently. In the eight cases, a tuberculin skin test was applied in five and was positive in four. This percentage is appreciably higher than in the general population but does not necessarily indicate a conclusive diagnosis in the individual case. In patients from known endemic areas, coccidioidin and histoplasmin skin tests should be performed in addition to tuberculin.

In the final analysis, exploration will be necessary in nearly all cases, especially for the surgeon who follows the philosophy that unilateral mediastinal masses should be subjected to early surgical investigation.

Should an obvious granuloma be removed when discovered at exploration? Technical difficulties have been encountered because of inflammatory adherence to surrounding structures. Minor accidents such as tearing of vessels and sacrifice of nerves have occurred. On the other hand, five of eight patients had definite symptoms referable to the mass. In one, the symptoms were severe and there was partial tracheal obstruction. This case was one of four whose mass was in a similar location, the right superior mediastinum somewhat posteriorly. While two had no symptoms, there was no assurance that difficulties would not develop should the mass remain. Since the majority of patients had symptoms, we believe that granulomas should be removed if exploration is done. Even if the majority, or all such granulomas, are proved tuberculous in the future, experience to date is reassuring in that no active tuberculous infections have developed postoperatively.

PATHOLOGY

The lesions described previously were without question chronic granulomas. All presented a similar picture of fibrosis, round cell infiltration, epithelioid cell reaction, multinucleated giant cells, and varying degree of caseation necrosis. Seven of the eight cases showed residual lymphoid tissue which means that in so far as the mediastinum is concerned, the "tumors" have been composed of a granulomatous infection in coalesced lymph nodes. In this, mediastinal granulomas apparently differ from similar masses in the lung.

TABLE I

SURGEON	AGE (YR.)	SEX	SYMPTOMS	MEDIASTINAL LOCATION	TUBERCULIN	OTHER SKIN TESTS	PREOPERATIVE DIAGNOSIS
Grace	32	F	Dyspnea, pain	L. Ant. Sup.	Positive	Not recorded	Dermoid
Blades and Dugan	50	M	Dyspnea	R. Post. Inf.	Not recorded	Not recorded	Neurogenic tumor
Paulson, No. 1	33	M	None	R. Post. Sup.	Positive	Not recorded	Mediastinal tumor
Paulson, No. 2	21	M	None	R. Post. Sup.	Positive	Not recorded	Mediastinal tumor
Authors, No. 1	27	F	Vague pressure, dysphagia	R. Post. Sup.	Not done	Not done	Dermoid
Authors, No. 2	19	M	None	L. Ant. Sup.	Negative	Cocciiodin negative	Dermoid
Authors, No. 3	22	M	Dyspnea, cough	L. Ant. Sup.	Not done	Not done	Bronchiogenic cyst
Authors, No. 4	34	F	Severe cough, dyspnea	R. Post. Sup.	Preoperative 0 (1 to 1000) Postoperative + (1 to 100)	Cocciiodin -0 Histoplasmin ++++	Mediastinal tumor, (?) aneurysm

Whether or not the infection is due to the tubercle bacillus cannot be answered with such certainty. In seven of the eight cases, neither tissue stains, cultures, nor guinea pig inoculations revealed tubercle bacilli. In one case, a few acid-fast bacilli were found by special staining methods. Pulmonary granulomas due to coccidiomycosis have been reported,^{5, 10, 11} presumably similar masses may occur in the mediastinum. *Histoplasma capsulatum* and syphilis may likewise produce caseating granulomas. As of the present moment sufficient clinical and laboratory investigations have not been done on these cases to prove conclusively the type of infection. When a granuloma is removed at operation, the tissue should be subjected to extensive cultural investigation and animal inoculation. Moersch and his co-workers¹⁰ have stressed the importance of these steps in the diagnosis of obscure pulmonary lesions; they are no less valuable in determining the cause of a mediastinal granuloma. In essence, we do not believe that unquestioned proof of the tuberculous nature of these granulomas is at hand.

In regard to the absence of acid-fast organisms, one must admit the evidence of Griffith¹² and Feldman and Baggenstoss¹³ whose investigations have suggested that viable virulent tubercle bacilli are rarely found in retrogressive, encapsulated lesions or in sclerotic lymph nodes.

SUMMARY

1. The available information on "tuberculomas" of the mediastinum has been summarized and four additional cases are reported in detail.

2. The possibilities of more accurate preoperative diagnosis are enumerated. As with other mediastinal masses, surgical exploration usually must be done in nearly all instances.

3. When a granuloma is diagnosed at the time of surgery, it is concluded that removal should be accomplished whenever possible.

4. The pathology has been discussed. The question is raised as to whether these granulomas are due to the tubercle bacillus or to other organisms. We believe that their tuberculous nature has not been unquestionably proved. Suggestions have been made as to future clinical and laboratory investigations which may aid in a more certain diagnosis.

REFERENCES

1. Burnam, C. F.: New Growths of the Mediastinum, *J. A. M. A.* 69: 989, 1917.
2. Haines, W. D.: Tumors of the Mediastinum, *Surg., Gynec. & Obst.* 20: 542, 1915.
3. Piazzzi-Martinni, V.: New Symptoms of Mediastinal Tumors, *Ann. di clin. med.* 5: 173, 1914. Cited in.¹
4. Heuer, George J.: Surgical Treatment of Tumors of the Mediastinum, *Ann. Surg.* 113: 357, 1941.
5. Grow, J. B., Bradford, M. D., and Mahon, H. W.: Exploratory Thoracotomy in the Management of Intrathoracic Disease, *J. THORACIC SURG.* 17: 480, 1948.
6. Grace, A. J.: Tuberculoma of the Mediastinum, *J. THORACIC SURG.* 12: 131, 1942.
7. Blades, B., and Dugan, D. J.: Tuberculoma of the Posterior Mediastinum, *Am. Rev. Tuberc.* 50: 41, 1944.
8. Paulson, Donald L.: Personal communication, Nov. 8, 1948.
9. Blades, B.: Mediastinal Tumors, *Ann. Surg.* 123: 749, 1946.
10. Moersch, H. J., Weed, L. A., and McDonald, J. R.: Bacterial Examination of Tissues Surgically Removed as an Aid in the Diagnosis of Diseases of the Chest, *Dis. Chest* 15: 125, 1949.

11. Greer, S. J., Forsee, J. H., and Mahon, H. W.: Surgical Management of Pulmonary Coccidiomycosis in Focalized Lesions, *J. THORACIC SURG.* 18: 591, 1949.
12. Griffith, A. S.: Types of Tubercle Bacilli in Human Tuberculosis, *J. Path. & Bact.* 32: 813, 1929.
13. Feldman, W. H., and Bagenstoss, A. H.: The Residual Infectivity of the Primary Complex of Tuberculosis, *Am. J. Path.* 14: 473, 1938.

DISCUSSION

DR. DONALD L. PAULSON, Dallas.—My purpose in discussing Dr. Samson's paper is to present two cases that he recorded in his presentation. (Slides.)

This represents a mediastinal mass located posteriorly in the mediastinum. This was found in a routine survey and was asymptomatic. Operation was advised because it was a mediastinal tumor of undetermined type. It was found at operation to be densely adherent to the esophagus and the pericardium in the region of the atrium, and was removed with some difficulty because of this dense adherence. It was a cystic mass which was opened during removal and a thick, green, creamy material exuded from the mass. It was entirely removed, however, except for the calcified upper pole which was densely adherent to the mediastinal structures and it was considered wiser to leave this behind. This patient did well for three weeks postoperatively but at that time developed a pleural effusion on the right, with low-grade fever. The fluid was aspirated, smears and cultures for tubercle bacilli were made; examination of the contents of the cystic mass were also made for tubercle bacilli, and none were found in either the fluid or the contents of the mass.

This patient eventually recovered but had a period of about six months of low-grade fever, in my opinion due to a pleurisy.

The next slide reveals a mass in the upper mediastinum. Extensive studies were made in an attempt to make a definite diagnosis. Bronchoscopy revealed no evidence of a bronchiogenic tumor. Exploration was advised and carried out. What was interpreted at that time to be a mediastinal tuberculoma was found within the mediastinum densely adherent to the azygos vein, the superior vena cava, the right innominate vein, and trachea. The mass was removed with a good deal of difficulty because of the dense adherence to these structures, and a few openings were made in these large veins which were easily closed.

Pathologic examination of both of these masses revealed a chronic granulomatous type of lymphadenitis consistent with tuberculosis.

As a result of this experience it was my opinion that if a diagnosis of mediastinal tuberculoma could be made clinically, perhaps one would be wiser not to advise exploration because of the dangers of spread of infection, as occurred in the first case, or damage to the surrounding mediastinal structures to which these granulomas are usually densely adherent. At the present time we have two other patients under observation, one of which is asymptomatic, which we have followed for three years. It appears to be undergoing progressive calcification. The second patient is an older woman who I presume has a tuberculoma densely adherent to the esophagus which is producing symptoms of dysphagia. In the second case surgery probably will be advisable. In my opinion surgery is indicated in these mediastinal granulomas, tuberculomas, perhaps, only if they are symptomatic or if the diagnosis is in doubt.

DR. A. J. GRACE, London, Ontario.—I should like to say a few words about my case, the first one listed in Dr. Samson's table. This patient was operated upon in 1940 and the case was reported in considerable detail in the December, 1942, issue of *THE JOURNAL OF THORACIC SURGERY*. (Slides.) These slides show the sharply demarcated tuberculoma lying anterior to the left lung root and apparently causing some pressure effects here to account for her apnoeic dyspnea. It will be noted that this tumor is of considerable size, being rather larger, I believe, than any of those in Dr. Samson's cases. The slide illustrating the section of the tumor, following removal, gives some idea of its nature and structure.

It may be noted that this patient first had symptoms from her tuberculoma following a blow on the left chest. Following that she had pain in the region of the left breast, periodic dyspnea, and a low-grade fever at times. Serial x-ray examinations showed some definite variation in size of the mass.

It may be noted that in this case several medical consultants were opposed to operation because of functional considerations in the clinical assessment of the case. Everything possible, including deep therapy, was tried first before she was turned over to me for operative treatment. Early operation is certainly advisable in most cases of undiagnosable mediastinal tumors, and it remains the only method of establishing a diagnosis as well as of curing the condition in many of these cases.

I have been talking to this patient quite recently and find that she remains well and free from any noteworthy chest symptoms nearly nine years following the operation noted previously.

DR. WARRINER WOODRUFF, Saranac Lake, N. Y.—I should like to ask about the definition of tuberculoma in a problem of this kind. These are certainly not similar to those we see in lung substance. Those in the lung are to all intents and purposes undrained tuberculous abscesses and are filled with an inspissated caseous material from which tubercle bacilli are recovered in large numbers. In not a single instance of Dr. Samson's cases were tubercle bacilli found, either in the cyst or in the sputum. I wonder if these cases should not be recorded in the literature under some other classification than tuberculoma.

We have recently had a case of a large posterior mediastinal cyst which I think would fit this description—a man, 60 years of age, with an old calcified lesion which looked as if he might have had tuberculosis years ago. It projected on both sides of the spine. So far as attachment was concerned, apparently it came from the region of the bifurcation of the trachea, although there was no communication. Pathologic studies are not complete, but I think this may be another case which would fit into Dr. Samson's description; but we did not call it tuberculoma.

DR. ALFRED GOLDMAN, Los Angeles.—I wish briefly to report an experience with a case of chronic granulomatous mass such as Dr. Samson showed, which had in it calcifications, and a sinus tract leading from the calcified area to the esophagus. This patient was sent to me as a possible carcinoma of the esophagus. Examination revealed a puckered red granulomatous mass involving one-half the circumference of the wall, and after going beyond the area one saw a few drops of pus from the opening in the esophagus. Biopsy showed chronic infection, and no acid fast bacilli were found. At operation glands were seen at the bifurcation of the trachea which contained about 15 c.c. of pus. The pus was sterile. Histologic examination of the mass was reported as nonspecific granuloma. The coccidioidin test was negative although the patient came from that area. The tuberculin test was not positive.

DR. SAMSON (Closing).—I agree with Dr. Paulson that if a diagnosis can be made many lesions give symptoms which are not diagnosed except by operation, and that course seems to be the best.

Dr. Woodruff did not see the paper, in which the word "tuberculoma" was in quotation marks. Personally I am very much undecided that they are tuberculomas, and as a matter of fact I suspect that many are not. Certainly they differ from the so-called tuberculoma of the lung.

Dr. Goldman's case is interesting. A granuloma usually is a specific entity if I remember correctly. While the etiology cannot always be determined I question whether we should use the term "nonspecific granuloma" in this generic sense.

ACCIDENTAL CYST OF THE CHEST WALL LINED WITH BRONCHIAL EPITHELIUM

REPORT OF A CASE

GRANT N. STEMMERMANN, M.D., AND ROBERT W. DUFFY, M.D.
NEW YORK, N. Y.

INTRODUCTION

THE FOLLOWING case is presented as an unusual occurrence, which illustrates the reaction of bronchial tissue to injury. No identical case was noted in a review of the literature.

CLINICAL HISTORY

V. C., a 32-year-old white man, entered Halloran Veterans Administration Hospital on May 5, 1947, complaining of a tender mass in the right side of the chest wall which first appeared in April, 1945, and increased in size until his admission. Past history revealed that he was struck by antiaircraft shell fragments while on a mission over Germany in August, 1943. His plane was shot down, and the patient received wounds of the right side of the chest. Following capture he was found to have a right pneumothorax, and surgical removal of metallic fragments was done through a short posterior chest incision at a German prisoner-of-war camp hospital. According to the patient, he developed an "acute pleurisy" in the right side of the chest postoperatively and was hospitalized for a period of six months. The wounds healed, and thereafter he was asymptomatic except for occasional pains in the right side of the chest. On April 25, 1945, at the time of his liberation, he first observed a small swelling on the right lateral side of the chest wall, which was slightly tender. In the spring of 1946 he observed an increase in size of the mass and complained of increasing tenderness over this area. He also complained of slight exertional dyspnea which continued until the date of admission. There was no accompanying cough or hemoptysis. Physical examination was negative except for a stellate scar 5 cm. in diameter 2 cm. to the right of the ninth dorsal vertebra, posteriorly. Present in the right side of the chest wall in the posterior axillary line was a lemon-sized mass which was hard, slightly tender, and firmly attached to the chest wall over the eighth to tenth ribs. It was not attached to the overlying skin. X-ray examination revealed the mass to contain a metallic foreign body within it (Fig. 1).

The mass was removed on May 13, 1947, under local field anesthesia. At operation the bands of the serratus anterior and latissimus dorsi muscles were found immediately overlying the mass and firmly attached to its thick, gray-white capsule, forming a portion of the outer wall of the cyst. The muscle bands were split and separated by sharp dissection to reveal a spherical mass beneath, which was tense and slightly fluctuant. The posterior aspect of the mass was separated with difficulty and was adherent to the underlying ribs and intercostal muscles by a dense avascular tissue which radiated in a fanlike fashion, fusing with the muscle fascia and periosteum. This was dissected and the mass removed, leaving the floor of the defect formed by the dense scar tissue previously described.

It was evident at the time of operation that the sequel to injury had been a marked tissue reaction to the presence of a foreign body. This involved not only the structures in the rib cage, but also the overlying muscles and fascia.

Gross Appearance.—The sac was tensely distended and measured 4.5 cm. in diameter. Its outer surface was pink in some aspects, gray in others (Fig. 2). When the sac was opened,

From the Laboratory and Surgical Services, Halloran Veterans Administration Hospital, Staten Island, N. Y.

Published with permission of the Chief Medical Director, Department of Medicine and Surgery, Veterans Administration, who assumes no responsibility for the opinions expressed or conclusions drawn by the authors.

Received for publication Oct. 13, 1948.



Fig. 1.—Roentgenogram, lateral view, showing metallic foreign body in superficial aspects of chest wall.

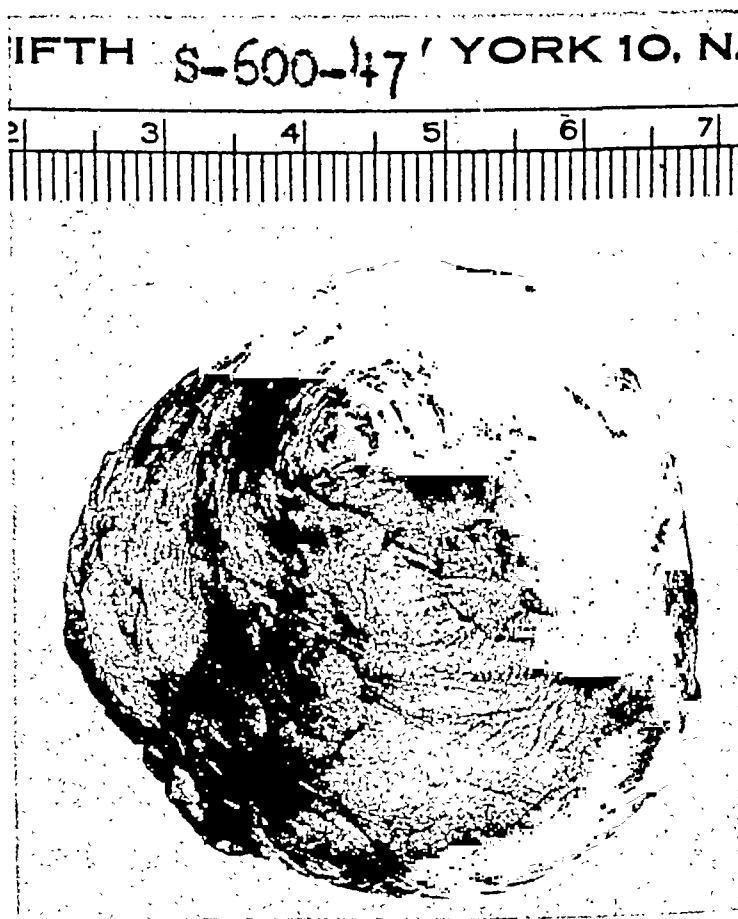


Fig. 2.—External appearance of foreign body cyst.



Fig. 3.—Photomicrograph of cyst lining showing stratified epithelium bordering vascular granulation tissue ($\times 100$).

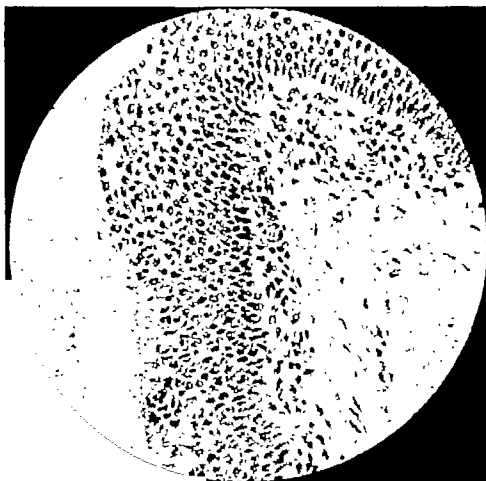


Fig. 4.—Photomicrograph of cyst lining near its transition from stratified squamous to ciliated columnar epithelium ($\times 400$).

it was found to contain approximately 15 c.c. of thick, tenacious, ginger-brown fluid. Contained within it also was a jagged sliver of steel shrapnel which measured 2 cm. by 6 mm. by 3 mm. It lay free within the lumen of the cyst. The wall of the sac measured 2 mm. in thickness, and its inner surface had a granular, red-brown appearance in some portions, and was lined by a smooth, gray membrane in others.

Microscopic Appearance.—Portions of the cyst wall were lined by stratified squamous epithelium (Fig. 3); other aspects were lined by a pseudostratified, ciliated columnar epithelium. Many of the cells in the latter portions had a goblet appearance (Figs. 4 and 5). Beneath the epithelium were dilated capillaries, histiocytes, plasma cells, and lymphocytes. In still other aspects of the wall the continuity of the stratified squamous epithelium was interrupted, and here the cyst wall was lined by a vascular granulation tissue composed of dilated capillaries, fibroblasts, collagenous fibers, many polymorphonuclear leucocytes, plasma cells,

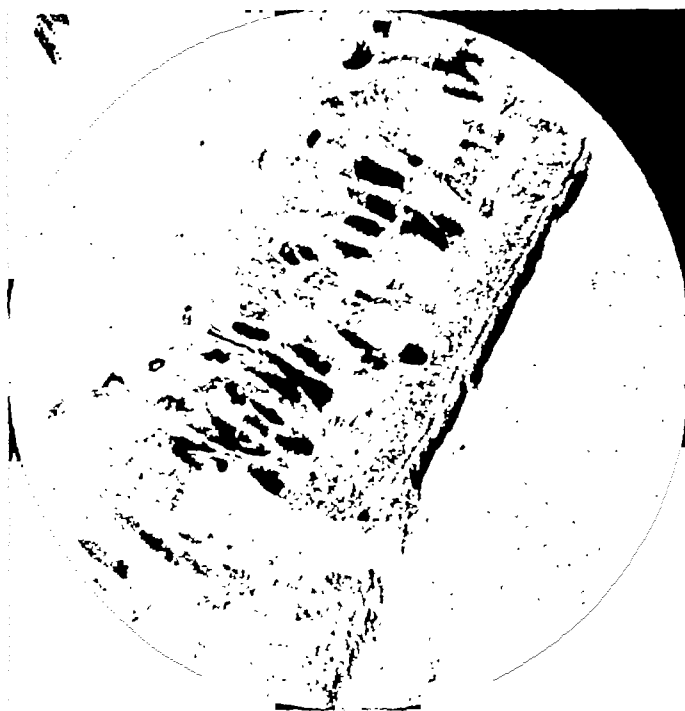


Fig. 5.—Photomicrograph of the mature ciliated columnar epithelium which lined portions of the cyst ($\times 950$).

occasional eosinophiles, and numerous macrophages heavily laden with brown pigment. This granulation tissue extended for a short distance beneath the adjacent stratified squamous epithelium. The outer portions of the cyst wall were composed of dense collagenous connective tissue which extended between and separated the muscle bundles adjacent to the outermost portions of the sac.

Subsequent sections from other portions of the circumference of the cyst wall revealed changes similar to those already described.

DISCUSSION

The manner in which the cyst came to be lined by a mature bronchial epithelium is of great interest. Two possibilities present themselves:

1. The cyst may represent a remnant of a bronchopleural-subcutaneous fistula. That such a fistula might have occurred is indicated by the presence of air in the affected pleural space after the original injury. As the tract healed

the lumen may have become narrowed by the developing scar tissue in its wall, so that eventually it was completely obliterated. Unfortunately the early history of the injury is not reliable, and whether or not it was followed by a frankly suppurative empyema cannot be ascertained. Such a sequence of events, however, has been reported in analogous situations. Auerbach and Green¹ have observed the epithelization of healed and healing tuberculous cavities. In such cases the new epithelium is either columnar or stratified squamous in type. These authors also quoted Pagel and Koch as having demonstrated the epithelization of pulmonary-cutaneous fistulas in cases of tuberculosis. Lisa and Rosenblatt² emphasized the re-epithelization of bronchi by ciliated columnar epithelium in cases of bronchiectasis. In all these situations one source of the new epithelial lining is from the healthy bronchial epithelium bordering the diseased areas. In the case of tuberculous cavities it is derived from the draining bronchi. In bronchiectasis small islands of intact epithelium may serve as an additional source of lining cells.

At the operation no such sinus tract was demonstrated, but the dense scar tissue which bound the cyst to the outer layers of the periosteum and intercostal muscles may represent the healed remnant of a fistulous tract.

2. The bronchial epithelium may have been carried to its definitive site by the jagged projectile. To accept this possibility it must be assumed that the journey was of such a nature that it did not destroy all of the transported tissue. Since only small numbers of healthy pavement cells are necessary to effect re-epithelization, this mechanism is a distinct possibility.^{3,4} The progress of the fragment through the chest was obviously a slow one. In contrast to rifle bullets, shell fragments are quickly spent. As the shell fragment collided with the ninth rib its progress was materially impeded. That it came to rest within the body also emphasizes its slow rate of travel.

Upon its arrival on the new site in the superficial aspects of the chest wall the epithelium may have come to line the existing tract or the space formed by the hematoma surrounding the foreign body.

It is well known that bronchial epithelium, when it has been subject to the insults of disease or injury, often becomes stratified squamous in type. Two factors served to make the new site a similarly unfavorable environment. The first of these was the foreign body reaction in the cyst wall. In addition, the active secretion of mucus by the epithelium considerably increased the pressure within the cyst. The presence of the stratified squamous epithelium in portions of the lining membrane represents, then, an expected reaction to injury. Miller⁵ quoted Baraban as believing the transition from ciliated columnar epithelium to stratified squamous epithelium to be due to low-grade inflammation which leads to loss of cilia and the appearance of mucus in the cells. Both Miller and Baraban agreed that in the lung coughing probably plays a prominent role in this transition. Condon⁶ believed that the stratified squamous epithelium which appears after injury to bronchial epithelium, as in mechanical trauma, chemical irritation, or disease is a pseudostratified form of normal respiratory epithelium. It represents not a metaplasia, but a protoplasia and is an early regenerative

form of epithelium. He stated further that the rapidity with which it is converted to ciliated columnar epithelium is dependent upon the state of the subepithelial tissue. In the presence of inflammation or irritation the conversion is a slow one. If Condon's hypothesis is a correct one, only a small amount of basement cells need have been the source of the epithelium which lined the cyst which we are reporting. From them the stratified squamous epithelium was developed, the transition to columnar epithelium representing the final step toward maturity.

That portions of the lining epithelium in our case were physiologically as well as anatomically mature is evidenced by the recent enlargement of the cyst during the year prior to its excision. The contents of the cyst were viscid, tenacious, and had a mucoid consistency. The ability of the epithelium to take root upon a damaged surface and to continue its specific function in a new location speaks for its resilient vitality.

SUMMARY

1. A foreign body cyst in the superficial tissues of the chest wall has been observed. It was lined by pseudostratified ciliated columnar epithelium, stratified squamous epithelium, and granulation tissue.

2. The cyst may have arisen as an accidental tissue transplant or may represent the remnant of a sealed-off bronchopleural subcutaneous fistula.

3. The reaction of the aberrant bronchial epithelium to injury mimics similar reactions in the diseased or injured lung.

REFERENCES

1. Auerbach, O., and Green, H.: The Pathology of Clinically Healed Tuberculous Cavities, *Am. Rev. Tuberc.* 42: 707-730, 1940.
2. Lisa, J. R., and Rosenblatt, M. B.: *Bronchiectasis*, New York, 1943, Oxford University Press.
3. Miller, W. S.: *The Lung*, ed. 2, Springfield, 1947, Charles C Thomas, Publisher.
4. Condon, W. B.: Regeneration of Tracheal and Bronchial Epithelium, *J. THORACIC SURG.* 11: 333-346, 1941.

ACTINOMYCES IN PUTRID EMPYEMA

COLEMAN B. RABIN, M.D., AND HENRY D. JANOWITZ, M.D.
NEW YORK, N. Y.

THE manner of development of pulmonary actinomycosis and our ideas concerning the pathogenicity of actinomyces for man require clarification.

Pulmonary actinomycosis may occur in three ways: (1) by direct extension from the abdomen through the diaphragm, (2) as a metastatic infection, (3) by inhalation.

The occurrence of infection of the lung through the diaphragm from an actinomycotic abscess of the liver perforating directly into the lung has been observed; this mode of infection of the lung has been well established but is not common. It is also well known that actinomyces may spread through the blood stream to produce hematogenous lesions in the spleen, kidneys, and bones, and it is possible for the lungs to become involved in the same way. However, in most cases of pulmonary actinomycosis, particularly in the earlier stages, the disease is found confined to the lungs, pleura, and chest wall, indicating that most cases of pulmonary actinomycosis have their origin in the airway, that is, that the infection is bronchogenous.

Actinomyces may conceivably be implanted in the lung by direct inhalation of the organisms from the air into the pulmonary alveoli, or they may be inhaled together with detritus or exudate from the mouth or throat in association with other organisms. This material may then become impacted in one of the smaller bronchi to produce infection of the lung. It is a peculiar fact that actinomyces, pathogenic for man, have never been found free in nature.^{3, 7} This, together with the apparent low pathogenicity of the organism, which will be discussed later, and the absence of evidence that the disease is contagious,⁷ make it extremely unlikely that infection of the lungs takes place by the direct inhalation of actinomyces floating free in the air.

A site of predilection for actinomycosis is the jaw; the infection usually arises in the gingiva, which is the common nidus for the actinomyces. Here the actinomyces are found together with other mouth organisms, many of which also are anaerobes. It has been a common observation, moreover, that actinomyces occur with great frequency in this location in persons either with no visible disease of the gums or with what appears to be simple chronic gingivitis or periodontoclasia (pyorrhea) without any evidence of actinomycosis as such. In addition, actinomyces have been found in the plugs within tonsillar crypts without evidence of actinomycosis of the tonsils.¹⁻⁵

In spite of the fact that the actinomyces isolated from such cases were anaerobic and adhered strictly to the cultural characteristics of the pathogenic form described by Israel and Wolff⁶ and known variously as *Actinomyces bovis*, *A. hominis*, and *A. israeli*, one can hardly consider them to be acting as pathogens

From the Medical Services and the Division for Thoracic Diseases, the Mount Sinai Hospital.

Received for publication Nov. 8, 1948.

in these cases. The difficulty that has been experienced in producing lesions in animals by the injection of these supposedly virulent organisms also indicates their low degree of infectiveness. It appears most probable that given an organism of such low-grade virulence, infection of the lung through the air passages must occur through the impaction in the smaller bronchi of particulate matter from the mouth or throat containing these organisms. If this material is not expectorated over a period of time, the likelihood of infection of the contiguous bronchus and the segment of lung that it supplies becomes apparent.

Under such circumstances it is rather to be expected that organisms carried down with the exudate or detritus from the mouth, together with the actinomyces, play a role in the ensuing infection. It has generally been assumed that bacteria found in the lungs in association with pulmonary actinomycosis are secondary invaders. But if the course of events postulated previously is accepted as the true pathogenesis of the disease, it must follow that these supposedly secondary invaders were present from the beginning, together with the actinomyces. Because of the difficulty which has attended the production of pulmonary actinomycosis by the intratracheal injection of pure cultures of actinomyces,⁵ it seems quite probable that these other nonspecific organisms play a part in the production of the infection by symbiosis.

A very similar course of events takes place in the production of putrid abscess of the lung. It is well known that the organisms responsible for this disease are not very virulent by themselves and that they are common inhabitants of the mouth or throat, where they may produce little or no disease. It has been amply demonstrated that this disease comes about from the inhalation of particulate material containing the anaerobes from the mouth or throat into the bronchi and that prolonged contact of the inhaled material with the contiguous bronchial mucous membranes leads to infection of the bronchus and the segment of lung which it supplies, to produce the localized area of gangrenous bronchopneumonia that leads to the putrid lung abscess.

A similar difficulty has been experienced in the attempts to produce putrid lung abscess by the injection of any one of the anaerobic organisms into the bronchial tree, and it has been generally concluded that in this disease symbiosis of the organisms is probably required for its production.⁹ No one will consider that the mixture of organisms found in acute putrid lung abscess represents a host of secondary invaders. They are all part of the original infection even though a good many of the organisms are comparatively avirulent and by themselves would not be responsible for infection.

A parallel between the pathogenesis of pulmonary actinomycosis and putrid lung abscess is found in the three cases reported in this paper. In each of them, there was a putrid empyema secondary to the rupture of a putrid lung abscess. These contained the usual flora that are encountered in lung abscess. In addition there was found in the pleural exudate of each of them the anaerobic *A. israeli* (*A. bovis*). In one case the pulmonary infection followed tooth extraction, and in the other two there was advanced periodontoclasia with purulent gingivitis. In one of these the actinomyces were searched for and found in the exudate about the gums.

CASE REPORTS

CASE 1 (M. S. H. No. 431467).—W. W., a 46-year-old lithographer, was admitted complaining of severe right-sided chest pain of four weeks' duration. His past history was not remarkable. Five months before admission he had had several molar teeth extracted under local anesthesia. His teeth were considered bad, he had had pyorrhea about them, and one or more teeth were broken in the process of extraction. Three months before admission the patient began to feel ill and developed a cough, which at first was non-productive, but later he coughed up a small amount of dark blood. During the month preceding admission to the hospital he developed severe, sharp pains in the right lower part of the chest and exertional dyspnea. He had occasional elevation of temperature, up to 100° F. on four occasions and experienced night sweats. An unsuccessful attempt to aspirate the right side of the chest had been made three days before admission.

Examination disclosed a well-developed and well-nourished, pale, white man in no acute respiratory distress. Temperature was 100.8° F., respirations were 24 per minute, and pulse rate was 96 per minute. He wore an upper partial denture, and he had moderate pyorrhea alveolaris. Several teeth were carious. His breath was not foul. The fingers showed slight clubbing. There was flatness over the posterior part of the right side of the chest up to the spine of the scapula; the breath sounds were suppressed over this area.

The hemoglobin was 82 per cent, the white blood cells numbered 12,000, and the differential count was normal. X-ray examination of the chest showed a small collection of fluid loculated over the posterior portion of the right upper lobe with marked thickening of the pleura over the entire right lung. Bronchoscopy was negative except for the presence of a small amount of thin, nonfoul secretion from the orifice of the right upper lobe bronchus. This did not recur after suction. Bronchography with Lipiodol revealed the branches of the lower lobe to be crowded together by the loculated fluid. Aspiration in the third intercostal space, paravertebrally, yielded a small amount of foul pus. A thoracotomy was performed, and a well-localized putrid empyema was found adjacent to the spine. The empyema space was shallow, and the visceral and parietal pleura were greatly thickened. In the mid-portion of the exposed visceral pleura there was an area of about one-half inch in diameter which was soft and presented a superficial erosion.

A smear of the putrid material obtained at operation revealed gram-negative bacilli, streptobacilli, and spirilla. Only *A. israeli* was grown from the empyema fluid.

The wound healed without incident. During two years of observation there was no recurrence of the symptoms, and the wound remained well healed. A film of the chest as early as one month after the operation showed no residual shadows.

CASE 2 (M. S. H. No. 444882).—L. C., a 35-year-old writer, was admitted with the chief complaint of pain in the right side of the chest of two weeks' duration and foul sputum for one week. His past history was noncontributory. His present illness began rather abruptly two weeks previously with severe pleuritic pain in the right posterior part of the chest. This was treated at another institution as a bronchopneumonia with sulfapyridine. One week after the onset, fever and foul sputum were noted, and he was then transferred to the Mount Sinai Hospital.

He was a well-developed and well-nourished, feverish young man in no acute respiratory distress. Temperature was 101.2° F., pulse rate was 92 per minute, and the respirations were 24 per minute. The dental consultant reported poor oral hygiene, calcareous deposits about the teeth, and periodontoclasia. His breath was not foul, and there was no clubbing of the fingers. There was dullness and diminution of breath sounds and fremitus in the right posterior part of the chest below the angle of the scapula. The entire right side of the chest was tender.

X-ray examination of the chest revealed a fusiform shadow in the region of the short fissure. Bronchoscopy revealed a small amount of thin pus with a peculiar sweetish odor issuing from the mesial and apical branches of the right lower lobe bronchus. Cultures of the sputum revealed hemolytic streptococci. During the following week he had a low-grade fever and the sputum had no odor. Repeated chest aspirations were negative. Finally,

aspiration in the sixth interspace just medial to the posterior border of the scapula yielded a small amount of thin, faintly odorous pus, and a thoracotomy was performed by resecting the eighth rib. The lung was covered with shaggy exudate containing a number of small collections of thin, turbid, foul pus. No pulmonary lesion was found.

The immediate convalescence was uneventful, the sputum disappeared, and the temperature promptly returned to normal. Bronchoscopy one week later was negative, and Lipiodol injected into the wound failed to enter the bronchial tree. Microscopic examination of the empyema wall revealed only acute and chronic inflammation. A culture was not made.

The patient was re-admitted five months later. He stated that the wound had continued to drain for two months and then closed. He had felt well until four days before admission, when he developed fever and pain in the scar. Physical examination revealed a fluctuant swelling in the center of the thoracotomy scar. This was incised and a cavity extending into the pleura, measuring about five by three inches, was found. No fistula could be discovered.

Microscopic section of the empyema wall revealed acute and chronic inflammation, with giant-cell formation. The wound healed shortly after discharge from the hospital, and the patient felt well for the following eight months. He then experienced chest pain and began to expectorate a small amount of yellowish, nonfoul sputum, which on culture, showed enterococci, *Streptococcus viridans* and *Staphylococcus albus* B.

Physical examination showed slight clubbing of the fingers. Once again the teeth were in poor condition. The scar was bulging and fluctuant. Incision of the swelling yielded a few ounces of foul pus. The cavity was smooth walled, and the operator was not certain whether it was intra- or extrapleural. No fistula could be demonstrated. Culture of the pus revealed *A. israeli*.

The patient returned to the hospital for further study one month later. Repeated examinations of the sputum, of the thin, mucoid secretion of the sinus tract, and smears and culture of material obtained by bronchoscopy revealed no actinomycetes. He was discharged with the wound healed. When last heard of through the follow-up clinic, three and one-half years later, the patient was well, had had no recurrence of symptoms, and the wound was well healed.

CASE 3 (M. S. H. No. 562568).—M. P., a 55-year-old white man, laborer, was admitted complaining of constant pain in the left side of the chest for three months. He had been hospitalized elsewhere eight months previously for cough and pain in the left side of the chest of one month's duration, and after one month of observation, was discharged. Three months prior to admission he noted severe pain on the left anterior and posterior side of the chest, shortness of breath, and a cough productive of small amounts of nonfoul whitish sputum. He had had profuse night sweats for two weeks.

He was a well-developed, well-nourished, dyspneic man, coughing up greenish, nonfoul sputum. Temperature was 103° F., the pulse rate was 120, and the respirations 24 per minute. Many teeth were missing, many of the remainder were carious, and there was a severe purulent gingivitis. The trachea was deviated to the right. There was dullness to flatness over the entire left lower lobe where breath sounds were absent, and there were diffuse wheezes throughout both lungs. The fingers were clubbed.

The hemoglobin was 56 per cent, and the white blood count numbered 17,100 per centimeter with 89 per cent polymorphonuclear leucocytes. Culture of the sputum yielded *Str. viridans*, *Str. hemolyticus* and *Micrococcus catarrhalis*. A chest film revealed an effusion in the left lower part of the chest, loculated anteriorly and lateral to the heart. There was an additional loculation posteriorly. A fluid level was present in the axillary portion of the chest at the height of the anterior portion of the third rib. The right side of the chest appeared normal.

Aspiration in the ninth space posteriorly yielded turbid, amber, odorless fluid which showed no organisms on gram stain, but a few small intracellular cocci were seen with methylene blue. Aspiration in the sixth interspace anterior to the angle of the scapula yielded foul air and pus. Operation disclosed a foul empyema overlying a pulmonary abscess about one inch in diameter, containing thick, putty-like, foul material. The abscess was unroofed.

Culture of the fluid from the empyema cavity, at operation, yielded *Staph. albus*, enterococcus and *A. israeli*. Section of the abscess wall revealed granulation tissue with purulent exudate, but no higher bacteria could be seen.

The patient received 400,000 units of penicillin daily for eleven days and 800,000 units daily for four weeks thereafter. He also received four blood transfusions, and the wound was revised twice. Two weeks after operation *A. israeli* was again cultured from the exudate in the wound. Radiotherapy was instituted, and a total of 1,200 roentgen units was administered. His temperature declined gradually over a period of four weeks and then remained normal. The wound was completely healed six weeks after operation. When the actinomyces were found in the pleural fluid, a search was made for these organisms in scrapings from the cavities of several of his teeth and from calcareous debris about the gingival margins. *A. israeli* was cultured from both locations.

COMMENT

The finding of pathogenic actinomyces, that is, the anaerobic *A. israeli*, in the pleural pus in three cases of perforated putrid lung abscess without the development of actinomycosis of the lung or chest wall is an indication of the low virulence of these organisms. In these cases the actinomyces did not behave differently from the general mixture of organisms involved in the putrid infection. In two of the cases the infection was cured by drainage alone. It is not known whether the infection would have cleared in the third case without the additional use of penicillin and radiotherapy. It is significant, however, that in the second case the infection cleared by drainage alone even though the actinomyces were present in the foul pus of an abscess in the wound sixteen months after the original thoracotomy.

The low virulence of actinomyces for the respiratory tract has also been pointed out by Kay,⁴ who found these organisms in the sputum of a large number of cases of nonspecific chronic suppurative disease of the lung without any evidence of pulmonary actinomycosis.

It is a common experience that patients with bronchiectasis at first have nonodorous sputum, and that foul odor appears in the later stages as the result of the contamination of the secretions in the bronchiectatic cavities by anaerobic bacteria from the mouth. The latter do not contribute materially to the further development of the pulmonary suppurative disease in the great majority of cases, but seem to act as saprophytes. It appears that the actinomyces found in the sputum by Kay behaved in a similar manner, implanting themselves from their original habitat in the mouth into the bronchiectatic secretion without producing significant bronchopulmonary disease.

A similar course of events, but with a different time sequence, appears to have taken place in the three cases here reported. It must be concluded that the actinomyces were carried down from the mouth together with a mixture of other bacteria, including anaerobes which were responsible for the lung abscess, while the actinomyces played a passive role.

Because of the low-grade pathogenicity of the actinomyces in respect to the lungs, it appears probable that other factors are required to produce pulmonary infection by these organisms, in addition to simple inhalation. Rosebury⁵ found that he could produce infection only by adding calcareous material to the culture of actinomyces, indicating that the impaction of particulate material together

with the actinomyces is a prerequisite for infection. Since this can rarely happen in the natural course of events without inhalation of other mouth bacteria, it must be concluded that these are also carried down into the bronchi at the time of the aspiration. These nonspecific bacteria certainly cause infection as easily as the actinomyces, and therefore, it seems most likely that nonspecific bacterial infection plays a role in the production of pulmonary actinomycosis, at least in its early stages.

Studies of the incidence of other types of fungi in the sputum in chronic pulmonary disease have shown that in the majority of cases the fungi act as secondary invaders, and are usually saprophytic.¹¹ This is such a frequent occurrence that a diagnosis of moniliasis, streptothricosis, leptothricosis, and aspergillosis is not tenable solely from the finding of these organisms in the sputum, unless other underlying diseases are entirely excluded.¹⁰⁻¹³ A similar conclusion must be drawn in relation to the finding of actinomyces in the sputum.⁸ Because of the three cases here reported the same reservation must be held even when actinomyces are found in pleural pus. A diagnosis of pulmonary actinomycosis is tenable only when fungi are found within the tissue itself.

SUMMARY

1. *A. israeli* was cultured in three cases of putrid empyema resulting from the perforation of a lung abscess. In one, this organism was also recovered from carious teeth.

2. None of the patients developed actinomycosis, although treatment in two cases consisted only of thoracotomy and drainage.

3. *A. israeli* is an organism of low-grade virulence, and is often present in the lung as a secondary invader or simply as a contaminant aspirated from the mouth and tonsils.

REFERENCES

1. Lord, F. T.: A Contribution to the Etiology of Actinomycosis. The Experimental Production of Actinomycosis in Guinea Pigs Inoculated With the Contents of Carious Teeth, Boston M. & S. J. 163: 82, 1910.
2. Lord, F. T.: The Etiology of Actinomycosis. The Presence of Actinomycetes in the Contents of Carious Teeth and the Tonsillar Crypts of Patients Without Actinomycosis, J. A. M. A. 55: 1261, 1910.
3. Lord, F. T., and Trevett, L. D.: The Pathogenesis of Actinomycosis. Recovery of Actinomyces-like Organisms From the Normal Mouth, J. Infect. Dis. 58: 115, 1936.
4. Naeslund, C.: Experimentelle studien über die etiologie und pathogenese der aktinomykose, Acta Path. & Microbiol. Scandinav. (Supp.) 6: 1, 1931.
5. Rosebury, T., Epps, L., and Clark, A.: A Study of the Isolation, Cultivation, and Pathogenicity of Actinomyces Israeli Recovered From the Human Mouth and From Actinomycosis in Man, J. Infect. Dis. 74: 131, 1944.
6. Wolff, F. M., and Israel, J.: Ueber Reincultur des Actinomyces und seine Uebertragbarkeit auf Thiere, Virchow's Arch. f. path. Anat. 126: 11, 1891.
7. Lord, F. T.: Actinomycosis, in R. L. Cecil: A Textbook of Medicine, Ed. 4, Philadelphia, 1937, W. B. Saunders Company, p. 354.
8. Kay, Earle B.: Bronchopulmonary Actinomycosis, Ann. Int. Med. 26: 581, 1947.
9. Cohen, John: The Bacteriology of Abscess of the Lung and Methods for Its Study, Arch. Surg. 24: 171, 1932.
10. Hamman, L.: Pneumococcosis, Am. Rev. Tuberc. 16: 575, 1927.
11. Thompson, S. T.: Note on Anaerobic Streptothrix Occurring in Sputa in Pulmonary Disease, Lancet 2: 1306, 1929.
12. Schneider, Leo V.: Primary Aspergillosis of the Lung, Am. Rev. Tuberc. 22: 267, 1930.
13. Stovall, W. D., and Greeley, Hugh P.: Bronchomycosis, J. A. M. A. 91: 1346, 1928.

USE OF THE POTTS-SMITH-GIBSON CLAMP FOR DIVISION OF PATENT DUCTUS ARTERIOSUS

WILLIAM S. CONKLIN, M.D., AND ELTON WATKINS, JR., M.D.
PORTLAND, ORE.

LIGATION of the patent ductus arteriosus with a single strand of nonabsorbable suture material is no longer an acceptable procedure. In a significant number of cases so managed there have been operative failures due to incomplete obliteration of the duct or its recanalization. Even with modifications of the ligation technique (use of multiple ligatures, transfixion sutures, reactive cellophane) there have been reports suggesting that recanalization may occur. The incidence of recanalization is difficult to evaluate because of the varying criteria for diagnosis and the lack of sufficient long-range follow-up. The presence of a postoperative murmur, per se, is not sufficient evidence that patency of the duct has persisted or recurred. Sometimes murmurs are audible immediately after a ductus has been completely divided. Changes in the contour of the pulmonary artery or pre-existing pulmonary artery dilatation with relative pulmonary insufficiency might account for such murmurs. The presence of other congenital anomalies must also be suspected. Consequently, a definite diagnosis of incomplete occlusion or recanalization should not be made on the basis of persistent murmur alone. If such a murmur is not typical of patent ductus in its location and character, if the murmur is present immediately following operation and does not subsequently change character, and if it is not associated with peripheral vascular signs of a systemic-pulmonary shunt, the diagnosis of recanalization requires further confirmation. However, Jones³ has noticed a decreased incidence of postoperative murmurs since he abandoned the use of multiple tape ligatures for division and suture of the ductus, and it is well known that any artery that has been ligated in continuity shows a tendency to recanalize. On such a theoretical basis and with limited clinical evidence, it may be suspected that division of the patent ductus arteriosus is the method of choice provided the surgical mortality is not increased.

Ductus division was carried out first by Touroff in 1940.² In his patient division became necessary to control hemorrhage which occurred while the ductus was being isolated preparatory to its ligation. Gross was the first surgeon to divide a patent ductus deliberately. His method was reported in 1944.⁴ This technique has proved eminently successful in the hands of a few surgeons. Four Crile artery forceps are applied across the isolated ductus. The two middle clamps are removed after dividing the ductus between them so that the small cuffs of vessel wall, that had been held between the jaws of the medial clamps, could be sutured with continuous rows of fine silk. In his original report Gross recommended that the jaws of each clamp be held closed by a tight rubber band

From The Department of Surgery, University of Oregon Medical School, the Surgical Services of the Doernbecher Memorial Hospital for Children, and the Matson Memorial Hospital for Diseases of the Chest.

Received for publication Nov. 29, 1948.

twisted about the handles of the clamp. In a more recent report,⁵ Gross stated that he had abandoned the use of rubber bands, closing each clamp to the first ratchet. Crafoord³ has applied clamps temporarily to the aorta above and below the ductus and has then divided the ductus while the aortic stream was completely interrupted.

Our experience with these two techniques of division has been limited. During our first attempt to divide the ductus by Gross's method, the Crile clamps, closed with rubber bands, slipped from position on either end of the ductus. Bleeding from the aorta and pulmonary artery was controlled with difficulty, but division and suture were completed and the immediate postoperative course was uncomplicated. Ten days after operation the patient developed classical signs of pulmonary embolism. There was no evident site for thrombus formation and, consequently, it was postulated that thrombosis had occurred at the site of ductus suture. The patient recovered and has done well, but in view of these complications and the relatively infrequent opportunity to perfect this method of division, we reverted to a technique of obliterating the duct with two ligatures of umbilical tape with an intermediate transfixion suture of heavy braided silk. Since April, 1947, we have used the method for division which will be described here and which has been mentioned in a previous publication.²

The high incidence of spinal cord lesions in dogs after complete acute clamping of the aorta, even of short duration, has deterred us from using Crafoord's procedure deliberately. Our only experience with clamping of the aorta during ductus division occurred at operation on a 9-year-old girl in 1946. The original plan was to perform double tape ligation, but during the dissection there was profuse hemorrhage from a tear in the posterior wall of the ductus. The aorta was clamped completely, proximal to the ductus, for periods of twenty-two minutes and thirteen minutes, with an interval of twenty-five minutes between, in order to control hemorrhage while the ends of the duct were sutured following its division. There was no evidence of leg paralysis after this prolonged period of aortic obstruction, and the postoperative course appeared uneventful, although a routine electrocardiogram on the tenth postoperative day showed T-wave inversion in the lateral precordial unipolar leads. This finding suggested a small area of myocardial ischemia although there was no evidence of infarction and the patient has continued to do well. The T-wave changes have disappeared. We are unable to explain such an occurrence and were unable to duplicate such an electrocardiographic change during clamping of the aorta in the dog.¹⁰

There are a number of factors that have contributed to the problem of devising an entirely satisfactory technique for division of a ductus. The average duct is a very short vessel with a large diameter. This makes simple division between ligatures extremely hazardous. The ligatures are likely to roll off the ends of the divided vessel, particularly at the aortic end where the large lumen is exposed to high systemic arterial pressure. Experience with pulmonary resections has demonstrated adequately that a single ligature, with a transfixion suture added to eliminate the danger of the ligature rolling off the vessel end, provides complete security for closure of the proximal end of a divided pulmonary artery. At its pulmonic end the wall of the duct has essentially the

same characteristics as that of the pulmonary artery. This end of the duct is exposed only to the relatively low blood pressure of the pulmonary artery after division of the ductus has been accomplished. Ligation and transfixion provide a satisfactory method for closure of the pulmonic end of the ductus unless the vessel is exceptionally large. The wall of the duct at its aortic end, on the other hand, is like the wall of the aorta; it is relatively thick and friable so that a simple encircling ligature tends to crush and to split the intima. The intimal damage which occurs with crushing of the aortic end of the ductus predisposes to recanalization when ligation techniques are used. Exposed to the high systemic arterial pressure, even a transfixion suture is likely to be dislodged from the aortic end of the duct and, should such a suture produce a small tear in the aortic wall, hemorrhage may become progressively severe and uncontrollable.

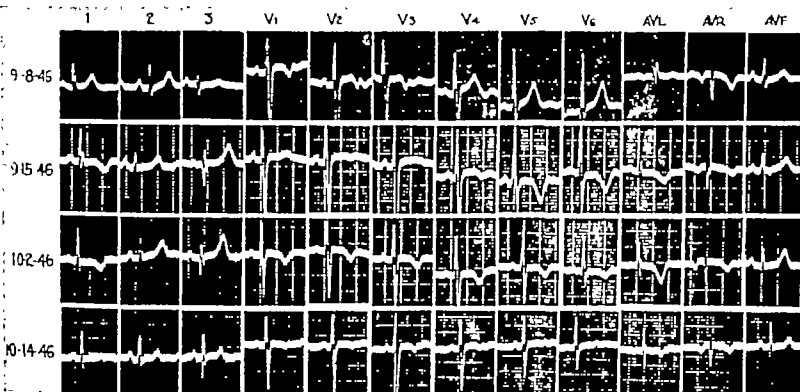


Fig. 1.—Electrocardiographic changes following complete clamping of the thoracic aorta distal to the left subclavian artery during division of patent ductus. The aorta was completely clamped for periods of twenty-two and thirteen minutes with an interval of twenty-five minutes between the periods of clamping. Preoperative electrocardiogram (Sept. 8, 1946) showed upright T waves in the unipolar precordial leads, V_4 , V_5 , and V_6 . Operation was performed on Sept. 9, 1946. The first postoperative tracing was recorded on the sixth postoperative day and showed inversion of the T waves in V_4 , V_5 , and V_6 . T-wave inversion regressed over a period of five weeks after operation.

The posteromedial wall of the ductus at its pulmonic end may be closely adherent to the left main bronchus. By the usual blind approach for dissection of this part of the ductus, it is often difficult to free sufficient length for division by ligature or suture techniques. The ductal wall is fragile here and is likely to be torn, especially if it has been weakened by superimposed bacterial infection.

Methods requiring the application of clamps to the ductus itself have many disadvantages: (a) danger of slipping or dislodgment of the clamps, (b) too small a cuff available for closure, (c) inclusion of thin ductal tissue in the aortic closure with the consequent danger of late aneurysm, (d) unsuitable for the very short ductus where four clamps cannot be applied, (e) trauma of the vessel wall close to the area of suture predisposing to rupture or aortopulmonary fistula.*

*A new clamp devised by Potts⁴ grasps the ductus with prongs without crushing it and avoids some of these disadvantages, particularly (a) and (c).

When the technique for creation of a side-to-side anastomosis between the pulmonary artery and aorta was reported by Potts, Smith, and Gibson⁷ it seemed that the aortic clamp described by them could be used as well to divide a ductus as to create one artificially. Consequently, the following procedure was developed which we feel offers the advantages of complete aortic obstruction without the dangers inherent in complete interruption of aortic blood flow.

OPERATIVE METHOD

In order to obtain adequate exposure and enough room for placing the Potts clamp on the aorta, a posterolateral incision is considered obligatory. The patient is placed on the operating table in the right lateral decubitus position. An endotracheal tube is used for the administration of cyclopropane and ether in a closed circle system. An ankle vein is cannulated routinely and a continuous slow infusion of dextrose solution maintained during the operation. Blood is available but is usually not required. The fourth rib is resected from behind its angle to the anterior axillary area and the pleural space is entered through the periosteal bed of the resected rib. It is particularly important that the rib resection be carried well medial to the angle of the rib posteriorly to facilitate manipulation of the aortic clamp. Occasionally it has been advisable to resect a small posterior segment of an adjoining rib to increase exposure.

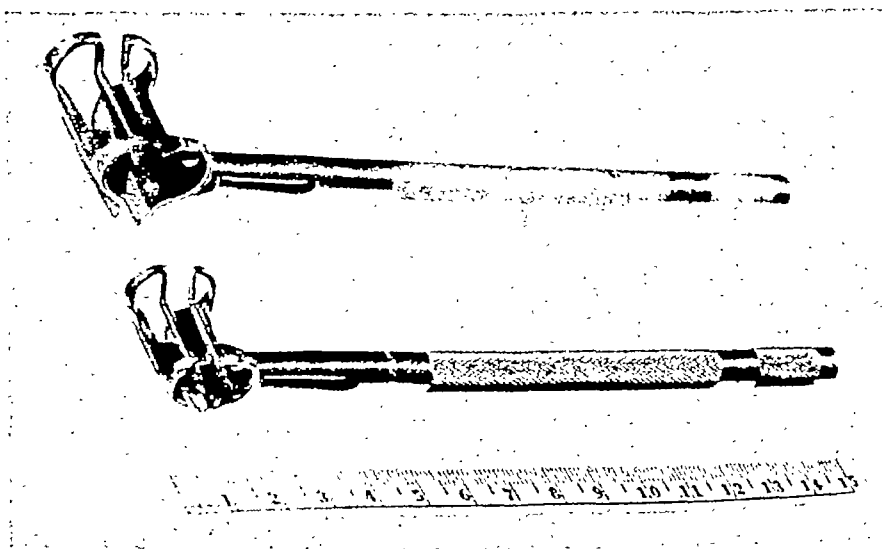


Fig. 2.—Aortic clamps of the type described by Potts, Smith, and Gibson. The lower clamp is the standard size described by them. The upper clamp was made locally for us. The diameter of the portion encompassing the aorta and the length of the jaw is one-third larger than the standard clamp dimensions.

The aortic clamp is of the type described by Potts and his associates.* The standard clamp used by Potts is too small to be used easily in any but the smallest patients considered for ductus operation. Our largest clamp was made locally and is one-third larger than the standard Potts clamp. The standard and larger clamps are shown in Fig. 2.

Mediastinal dissection is started by incising the pleura over the first part of the descending aorta from the origin of the left subclavian artery to a point about 2 cm. distal to the ductus. A side extension of this incision, passing directly over the ductus, may prove desirable later in the dissection. The aorta is exposed by subpleural dissection and the segment opposite the ductus orifice is mobilized. It may be necessary to divide one or more intercostal arteries and smaller branches of the aorta passing to the mediastinum.

*Available from V. Mueller & Company, Chicago, Ill.

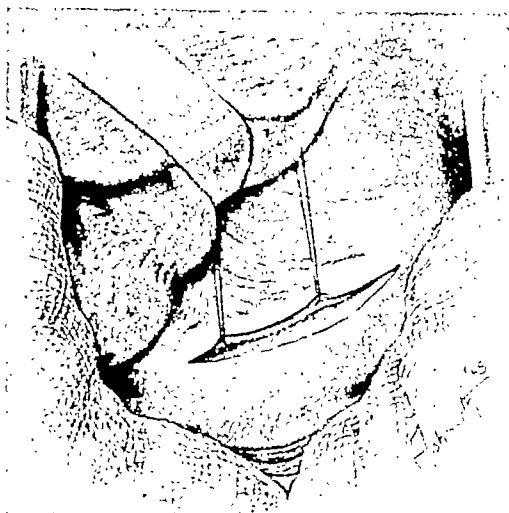


Fig. 3.—Incision of the mediastinal pleura over the first part of the descending aorta. This incision is more posterior than the usual incision for ductus ligation.

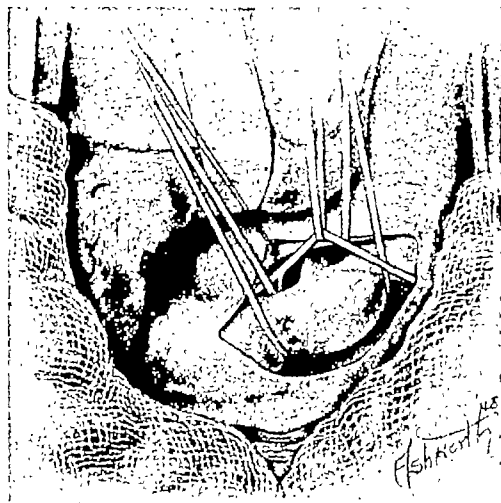


Fig. 4.—Dissection of the aorta and ductus is completed. Umbilical tapes are placed around the aorta above and below the ductus. The vagus nerve is retracted anteriorly showing the recurrent laryngeal nerve passing medial to the ductus. One intercostal artery has been divided.

Care must be taken that such vessels are not sheared off close to the aorta. Division between ligatures is recommended rather than division between clamps. One must avoid injury to the thoracic duct while dissecting the aorta away from the thoracic vertebrae. If this vessel is traumatized, ligation is essential to prevent chylothorax.

When the aorta is adequately mobilized, strips of umbilical tape are passed around it above and below the ductus. Gentle traction with these tapes exposes the posterior aspect of the aortic end of the ductus so that dissection can be completed preparatory to placing the aortic clamp. The recurrent laryngeal nerve is mobilized so that it will not be included in the jaws of the aortic clamp. The Potts clamp is then placed around the aorta and

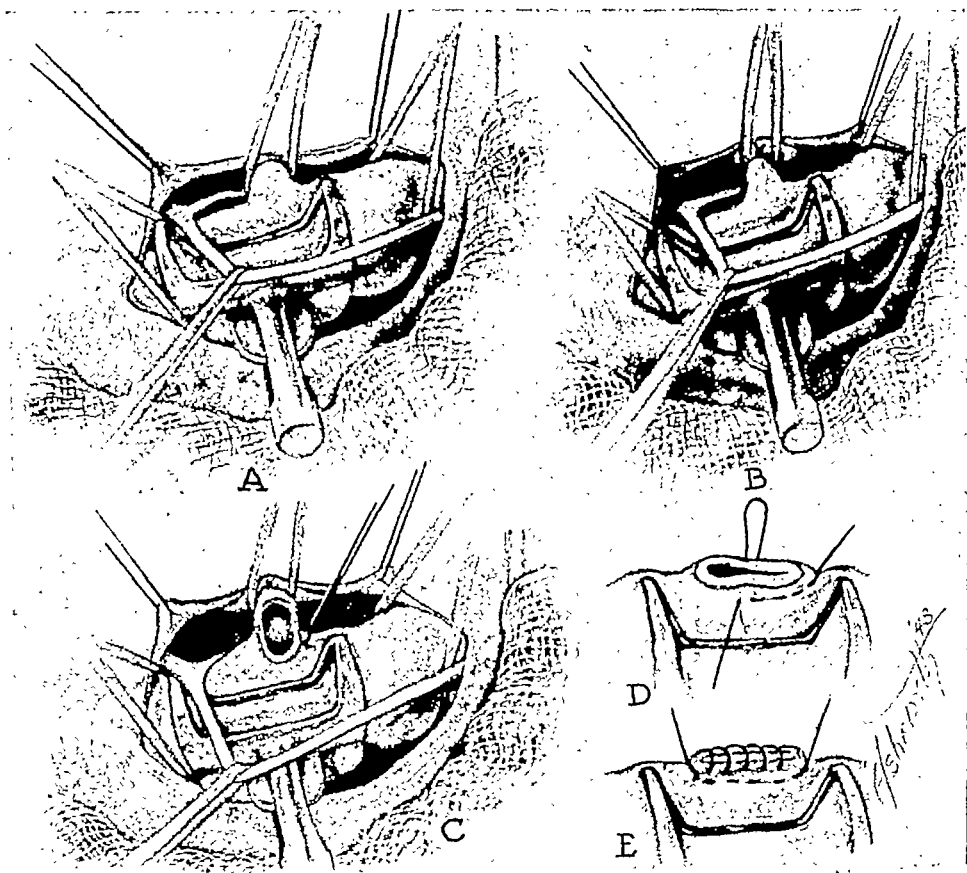


Fig. 5.—Details of the division and suture.

A, The clamp isolates the ductus from aortic blood flow but allows blood to pass into the descending aorta.

B, The pulmonary end of the ductus is ligated and transfixed as close to the pulmonary artery as possible.

C, The initial artery stitch is anchored at the upper angle of the aortic end of the ductus. The ductus is then divided. For purposes of clarity in this drawing, the vagus nerve is retracted to the aortic side of the ductus incision although in practice it is necessary to retract it toward the ligature on the pulmonary end of the ductus. Less tension is placed upon the vagus nerve than is shown.

D, The aortic end of the divided ductus is repaired with a continuous mattress suture of artery silk. Intima is approximated to intima.

E, The mattress suture is overlaid with a continuous glover's stitch.

slowly closed to occlude the aortic end of the ductus. The encircling tapes may be used to hold the aorta in position within the clamp during its closure. As soon as the clamp is completely closed the continuous thrill over the pulmonary artery and lung is eliminated. Appearance of a systolic thrill over the aorta distal to the clamp indicates that blood flow through the aorta has not been completely interrupted. The blood pressure in the arms may show a variable rise.

When the anesthetist has determined that latent cyanosis has not become evident as a result of eliminating blood flow through the ductus, isolation of the duct is completed. The dissection is greatly facilitated by early mobilization of the aorta and occlusion of the aortic end of the ductus. After elevating the aorta carefully by means of the clamp, the thin posterior wall of the ductus can be freed from the left main bronchus under direct vision.

The pulmonic end of the ductus is ligated with heavy braided silk and transected with medium braided silk. The ductus is then divided between the pulmonic ligature and the aortic clamp. The vagus and recurrent laryngeal nerves should be retracted to the pulmonic side of the point of sectioning to avoid injury. These nerves are then allowed to fall between the divided ends of the duct where they will not interfere with closure of the aortic end. The aortic clamp is elevated and rotated to present the divided end of the duct. This is repaired with a continuous horizontal mattress suture of 00000 Deknatel on a curved atraumatic needle which approximates intima to intima. The first stitch of this

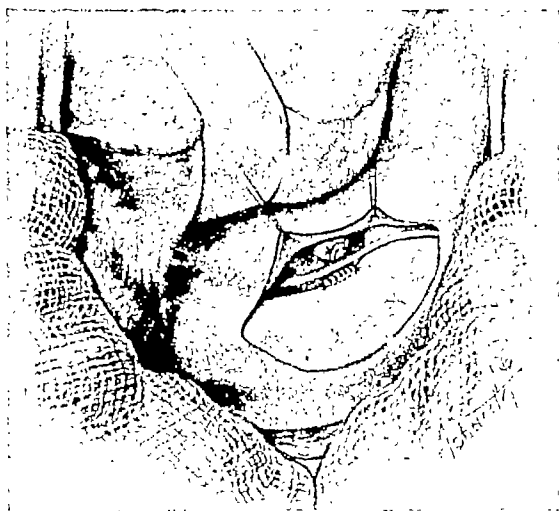


Fig. 6.—Operative field after division.

suture is placed above the proximal angle of the proposed line of division and close to the proximal jaw of the Potts clamp. By placing and tying this stitch before the duct is divided, bleeding which might occur from leakage around the proximal portion of the clamp is obviated. The running mattress suture should conform to the curve of the aortic wall avoiding creation of an appreciable pouch in the aortic wall. The closure is reinforced with a running glover's stitch over the divided end of the duct and in the cuff created by the mattress stitch. The aortic clamp is released slowly. If appreciable bleeding occurs, the clamp can be retightened and additional sutures placed. Oozing through the points of needle puncture may occur but will subside spontaneously.

During the operation the anesthetist inflates the collapsed left lung at intervals. Warm isotonic saline solution is used to keep the pleural surfaces moist. Unless marked tachycardia is present, 3 to 5 c.c. of 1 per cent procaine solution are injected about the left vagus nerve early in the operation. Following a final irrigation of the pleural cavity with

warm isotonic saline solution, the incision in the mediastinal pleura is loosely approximated with interrupted stitches and the chest wall closed in layers while lung expansion is maintained with water suction through a catheter placed in the wound. This catheter is removed during the closure of the skin. No drains are used.

We have used this method for division of the patent ductus arteriosus in eleven patients. Early in the series one patient succumbed at the time of operation as a result of hemorrhage. The hemorrhage is attributable to faulty technique. Lacking a Potts aortic clamp of sufficient size to completely isolate the aortic end of the ductus, the lip of a very large and dilated aorta, which bulged from between the jaws of the clamp, was occluded by compressing it with a loop of umbilical tape drawn tightly against the jaws. As a result of this ill-advised maneuver the aortic wall was split, the tear extending rapidly in a proximal direction so that all attempts to suture it were of no avail. We would like to stress the importance of using a clamp of appropriate size in each case, believing that the tragic outcome in this case might thereby have been avoided. No mishap has occurred during or following surgery in the other ten patients and all have remained well. Six of these patients had a soft systolic murmur over the left upper chest following operation. There has been no change in the character of these murmurs and there have been no other physical findings suggesting recanalization. In one of the six patients an isolated stenosis of the left pulmonary artery was found just distal to its ductal orifice and this may be the source of the persistent murmur. There has been no apparent correlation between postoperative murmurs and anatomic evidence of pulmonary artery enlargement.*

DISCUSSION

Use of the Potts-Smith clamp for division of patent ductus arteriosus provides certain advantages despite the more extensive dissection of mediastinal structures required. Mobilization of the aorta has allowed more freedom in the development of deep tissue planes about the ductus and in freeing the fibrous bands attached to the posterior wall of the ductus. During dissection of the thin-walled duct a measure of satisfaction can be taken from the knowledge that the aorta is mobilized for temporary clamping in case of hemorrhage. After the aortic clamp is closed, the dissection can be completed without the danger of spontaneous rupture from full aortic pressure within the unsupported walls of a thin ductus, one whose walls are damaged by bacterial endarteritis, a ductus

*Since preparation of this manuscript, ten additional patients have had ductus division by this technique. One more patient died of hemorrhage. In this case operative findings suggested marked pulmonary hypertension, though pulmonary artery pressure was not measured. The left pulmonary artery and the ductus were both enormous in caliber; nevertheless, placement of the aortic clamp and isolation of the duct were readily accomplished. While ligating and transfixing the pulmonary end of the duct, prior to dividing it, the pulmonary artery tore and the patient became exsanguinated before intrapericardial isolation of the pulmonary artery, and its temporary occlusion with a Blalock clamp, could be completed. While ligation and transfixion will control the pulmonary end of the duct satisfactorily in most instances, it is evident that a vessel of large caliber will be crushed and subject to tear when obliterated by a surrounding ligature, especially when ligation is performed close to junction with a large tense vessel like the pulmonary artery in this case. Plastic suture of the pulmonary end of the duct, as well as the aortic, is advisable in some cases. Potts has devised ductus clamps¹ with prongs which grasp and occlude the vessel without crushing and without danger of slipping. In our most recent case, with a large short ductus, both ends of the duct were sutured using a Potts ductus clamp on the pulmonary end and a Blalock modification of the Potts aortic clamp¹ on the aortic end during division and suture. The operation proceeded very smoothly. The latter clamp, being narrower at its back than the original design of Potts, is more readily fitted to the aortic end of the duct, particularly when the origin of the left subclavian artery is close to that of the ductus arteriosus.

with aneurysmal dilatation. The period of temporary clamping while the dissection is completed allows considerable time for the development of latent cyanosis in the patient with unsuspected pulmonic stenosis, although we have seen no case of this type where the diagnosis could not be made without operation. With a clamp of the Potts design, there is little danger of slipping since the aorta is encircled. Any pulsation is transmitted equally to the encompassing jaws. Inspection of aortic segments occluded by the clamp at operation and in the animal laboratory reveals no evidence of aortic damage. Occasionally a small subadventitial hemorrhage will be seen along the site of application, but there has been no evidence of trauma to the media or intima.

Because of the peculiar trough shape of the portion of the clamp isolating the ductus, a sizable segment of aorta is excluded for suture. Consequently, a double row of sutures can readily be placed along the aortic end of the ductus at its junction with the aorta. The contour of the clamp should also allow its use for division of very short ductuses, where the technique of clamping the ductus proper cannot be used. A portion of aortic intima and ductal lumen is available for inspection. Evidence of endarteritis may be demonstrable and the sutures may be placed accurately in the aortic wall proper. Both ends of the ductus can be repaired in such a way that no significant vascular pouch will remain at the side of the aorta or pulmonary artery.

Experience with side-to-side anastomosis of the aorta to the pulmonary artery indicates that great care must be taken in suturing the aorta to avoid tearing of the friable vessel wall. Of all the great vessels, the aorta seems particularly subject to tearing, and to us this hazard has seemed greater in suturing longitudinal incisions than transverse incisions, as in the resection of aortic coarctation.

The major disadvantage of this method has been that it is sometimes difficult to place the clamp so that its jaws completely encircle the aorta proximal to the ductus. This problem is encountered when the origin of the ductus is more proximal than usual so that the clamp must be placed at a point where the curvature of the aortic arch is prominent. Placement of the clamp is particularly difficult when fusiform dilatation of the aorta extends proximal to the ductus, when the aorta is large and relatively inelastic as in older age groups, and when the origin of the left subclavian artery is close to that of the ductus. Careful dissection of the deeper surfaces of the aorta above the ductus minimizes this disadvantage, but occasionally, even with adequate dissection, considerable manipulation has been required to place the clamp in position. A clamp that is adequate in size to encircle the dilated aorta or subclavian origin and has been modified to fit the medial curve of the aortic arch should eliminate this difficulty.* Death of our second patient is ascribed to the use of a clamp which was too small to encircle a dilated aorta. The excessive manipulation required produced tear-

*Since preparation of this manuscript, the article of Freeman, Leeds, and Gardner (Surgery 28: 103, 1949) came to our attention. They have modified the Potts aortic clamp to overcome certain difficulties encountered in its use for ductus division. The Bialock modification, as mentioned in a previous footnote, also appears to have definite advantages.

ing of the aorta. However, with increasing experience, we feel that the increased control and exposure available during dissection of the ductus proper will compensate for any additional risk.

SUMMARY

1. A technique for division of patent ductus arteriosus is presented. The Potts-Smith-Gibson clamp, designed for the creation of aortopulmonary shunt by suture, has been adapted to the reverse procedure, division of patent ductus arteriosus.

2. Application of the procedure is reported.

We are indebted to Dr. Marvin Schwartz for cardiology consultation and to Dr. Selma Hyman for radiologic interpretations.

REFERENCES

1. Bing, J. R., Handelsman, J. C., Campbell, J. A., Griswold, H. E., and Blalock, A.: *Ann. Surg.* 128: 803, 1948.
2. Conklin, W. S.: *Dis. Chest.* 14: 317, 1948.
3. Crafoord, C., and Nylin, G.: *J. THORACIC SURG.* 14: 347, 1945.
4. Gross, R. E.: *Surg., Gynec. & Obst.* 78: 36, 1944.
5. Gross, R. E.: *J. THORACIC SURG.* 16: 314, 1947.
6. Jones, J. C.: *J. THORACIC SURG.* 16: 305, 1947.
7. Potts, W. J., Smith, S., and Gibson, S.: *J. A. M. A.* 132: 627, 1946.
8. Potts, W. J.: *Quart. Bull., Northwestern Univ. M. School* 22: 321, 1948.
9. Touroff, A. W.: *J. A. M. A.* 115: 270, 1940.
10. Watkins, E.: *Surgery* 22: 530, 1947.

THE ROLE OF ESOPHAGEAL MOTILITY IN THE SURGICAL TREATMENT OF MEGA-ESOPHAGUS

J. DARIN PUPPEL, M.D.
COLUMBUS, OHIO

THE ACTUAL underlying physiopathology of mega-esophagus is still a matter of uncertainty in spite of much work done and many theories set forth in an attempt to understand the problem. There have existed three great schools of thought regarding the pathologic physiology of mega-esophagus. According to the theory of achalasia¹⁻⁵ which has been generally accepted by many modern workers,⁶⁻⁸ mega-esophagus is due to a lack of relaxation of the cardiac sphincter which normally occurs⁹ when each peristaltic wave conveying food through the esophagus reaches it. In support of this theory are the findings of Rake,¹⁰ Cameron,¹¹ Beattie,¹² Etzel,¹³ Lendrum,¹⁴ and others who have demonstrated degenerative changes in the ganglion cells of Auerbach's (myenteric) plexus. When the myenteric plexus is partially or wholly destroyed, such as was done by Tamiya and Tamura,¹⁵ the esophagus becomes irregularly active and later quiet, and the cardia fails to open normally in response to an oncoming bolus of food. On the basis of experimental work and as a result of clinical and post-mortem observations, Etzel¹⁶ came to the conclusion that the degeneration in human beings is due to a chronic vitamin B₁ deficiency. The achalasia theory explains that the disease is produced by disturbance in the nerve-muscle mechanism of the cardia. This is dependent upon degenerative changes in the vagal nerve supply which result in an inability of the circular muscle to relax due to preponderance of sympathetic control.

Knight¹⁷ has concluded from his experiments on the cat that there exists a true functional esophageal sphincter at the cardia. A condition resembling cardiac achalasia¹⁷⁻¹⁹ can be produced in some animals by complete or partial bilateral vagal section, which is relieved by subsequent division of the appropriate sympathetic fibers.¹⁷ Furthermore, simultaneous division of the sympathetic nerves prevented this condition.¹⁷ Following the double denervation there occurs eventually a normal movement of food after temporary disturbances.¹⁷ On the basis of these experimental findings division of the sympathetic nerves has been carried out for cardiac achalasia in man with some promising results²⁰⁻²³ but certainly not always with relief to the patient,²⁴ so that it seems that it is still better to dilate the esophagus and later, as necessary, to cut the sphincter in a manner such as by esophagogastrostomy of the Finney type. There is a wide origin of the sympathetic fibers which supply the esophagus,^{25, 26} so that some of the failures of sympathectomy may have been due definitely to incomplete sympathetic denervation of the affected esophagus.

Among the many other theories, Mikulicz²⁷ and Huss,²⁸ as well as others, popularly contended that the dilatation of the esophagus occurred secondary to a true spasm of the cardia. Carlson, Boyd, and Pearcey²⁹ confirmed and extended

From the Department of Research Surgery of The Ohio State University.
Aided by a grant from the Conely Fund for Research of The Ohio State University.
Received for publication Nov. 29, 1948.

Openchowski's³⁰ observations on the reflex control of the cardia and lower esophagus, which laid the groundwork for the possible existence of cases of clinically true cardiospasm. However, because of the poor results obtained by various surgeons³¹⁻³⁴ after bilateral vagotomy for idiopathic mega-esophagus, it was generally agreed that this procedure is not justified because its rationale is apparently based on a false premise.

The third group, including Sauerbruch and von Hacker,³⁵ Jackson,³⁶ and Hill³⁷ among others, widely advocated a phrenospasm as the underlying cause of the condition. However, the observation of obstruction above and below the diaphragmatic crura appears to rule out the possibility of their malfunction as a cause of the symptomatology.³⁸ Because gastrointestinal difficulties sometime follow unilateral phrenicectomy, Ballon, Wilson, Singer, and Graham³⁹ investigated the condition of the esophagus and stomach after this operation. In the two cases of mega-esophagus to be reported here, the obstruction also occurred above the diaphragmatic crura. Furthermore, in spite of arrest of movement of the left leaf of the diaphragm following phrenic crush during transthoracic exploration, the esophageal hiatus, which was normal and more than adequate in size to transmit the constricted cardiac end of the esophagus, did not increase in size as might be expected if the so-called cardiospasm were due to a phrenospasm. Division of the diaphragmatic crura^{40, 41} and phrenicoexeresis⁴² were accomplished by many but with unsatisfactory effects, so that these procedures have been generally discontinued as independent methods of treatment of mega-esophagus.

To the surgeon the problem of the underlying pathologic physiology of achalasia is a matter of importance. Many surgeons, including Ochsner and DeBakey,⁴³ have noted that even after adequate esophagogastrostomy the esophagus frequently does not return to its normal size, as the stomach returns to a normal size following the surgical relief of hypertrophic pyloric stenosis. Clagett and his associates⁴⁴ stated that although good functional results are obtained, retrogression in size of the dilated esophagus after esophagogastrostomy should not be expected. The problem of the underlying physiopathology of achalasia is further important since, we are convinced, the late postoperative clinical end results, even though good, are frequently not as pleasing when the esophagus remains atonic and dilated.

Recently we had an unusual opportunity to study this problem since we had noted similar phenomena following adequate esophagogastrostomy, as determined by satisfactory relief of symptomatology, by the barium swallow with Trendelenburg position test, and by postoperative esophagoscopy. Because methods of determining the functional behavior of various parts of the esophagus, heretofore, have been so equivocal and inadequate, we have felt that esophageal motility studies by the balloon and kymograph method may be of help in solving this problem. We have made esophageal motility studies of three normal persons under varying conditions and of two patients following esophagogastrostomy in the treatment of esophageal achalasia. These data indicate that the intrinsic motility of mega-esophagus varies greatly, being often irregular, uncoordinated,

and ineffective, and that it may be entirely absent, thus disclosing that a disturbed peristalsis or even total motor paralysis of the whole esophagus is present. We now wish to present the clinical and esophageal motility studies together with a detailed correlation between these two phases of the work.

EXPERIMENTAL METHODS

An easily distensible and markedly strong balloon was attached to an Einhorn tube in the usual manner. In order to determine accurately by fluoroscopy and by x-ray film studies (Fig. 1) the position of this balloon in the esophagus, we coated its inner lining with a radiopaque material, Rugar.



Fig. 1.—The position of the Rugar-lined balloon in the esophagus was easily determined by fluoroscopy and x-ray film study. In this patient (O. P.) with achalasia, as for normal persons, the balloon could be inflated only slightly without resulting in pain. A deglutition effect, which was less than normal, was almost constantly present, as measured by an almost continuous pull on the Einhorn tube. Esophageal fluid collected over the top of the balloon when the latter was held in one position during an experiment.

After placing the balloon in the desired position, the tube was connected to a bromoform-water manometer (Fig. 2). The balloon was never inflated to such an extent that it acted as an obstructing agent but rather as the usual bolus of solid food. In order to keep the distance from the incisor teeth to the balloon constant during any one experiment it was necessary to anchor the Einhorn tube securely to the patient's face with adhesive. This was done to counteract the constant pull on the Einhorn tube due to the constant attempt, by the muscular contraction and peristalsis of the normal esophagus, to force the balloon onward into the stomach. Observations were made in each person at three levels of the esophagus; the upper esophagus, above the aortic arch; the middle of the esophagus; and the lower esophagus, just above the diaphragm.

At the beginning of each experiment the balloon was inflated to the desired pressure of bromoform. The time and motility were recorded by a slowly revolving long kymograph. The inkwriters for the electric automatic timer, which recorded every ten seconds, and for the

manometer (Fig. 2) were composed of Monel metal, were sensitive, and recorded beautifully in ink* the time and the motor activity of any part of the esophagogastrintestinal tract.⁴⁵ The apparatus (Fig. 2) is easily assembled and much less fragile than former recording apparatus. With this apparatus the recording of motility is as easy as the recording of the basal rate of metabolism by the Benedict-Roth apparatus.

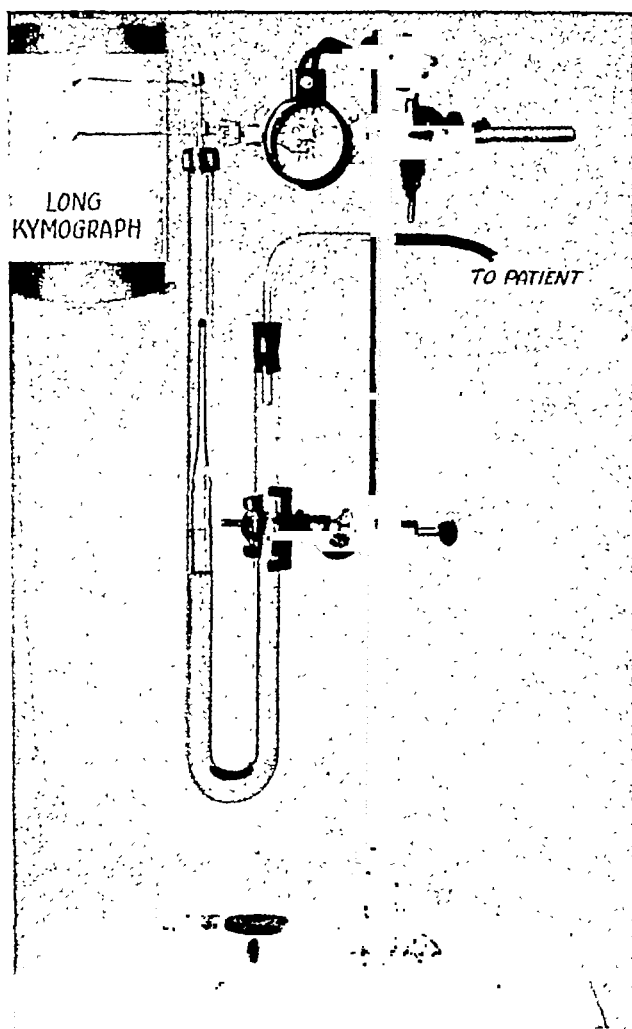


Fig. 2.—Apparatus useful for motility studies of the digestive tract. The inkwriters for the electric automatic timer and for the manometer are composed of Monel metal. The apparatus is easily assembled and much less fragile than former motility recording apparatus.

Each experiment was made in the postabsorptive state under basal conditions. Information obtained by barium swallow and by the observed clinical effects upon the inflated balloon was complementary to that obtained from the kymograms. The data considered together gave adequate conceptions of the action of the musculature of the esophagus.

ESOPHAGEAL MOTILITY STUDIES OF MEGA-ESOPHAGUS

The clinical, radiologic, and esophagoscopy progress, as well as the esophageal motility of two patients with mega-esophagus, was studied during a period of from one and one-half to

*The paper and ink were obtained from Warren E. Collins, Inc., Boston, Mass.

two and one-half years following esophagogastronomy of the Finney type. The protocols may be reviewed briefly as follows:

Protocol of O. P., No. 460072.—The patient, a white schoolgirl, 17 years of age, first became aware of difficulty in swallowing in 1937, at 9 years of age. She was known as "graveyard" at 15 years of age because she was almost five feet tall and weighed only 50 pounds. Because of her symptoms she followed a very poor and capricious dietary regimen. She had had carious teeth for years.

Physical examination disclosed the patient to be well developed but emaciated and ansthenic. She was five feet two inches in height but weighed only 80 pounds. There were many markedly carious teeth. Most of the reflexes were moderately hyperactive.

The usual laboratory tests were negative. Fluoroscopy as well as x-ray film studies (Fig. 3, A) of the esophagus during barium swallow on Oct. 4, 1943, revealed typical evidence of achalasia. Radiography of the colon revealed it to be atonic with a tendency to megacolon.⁴⁷⁻⁴⁹ Esophagoscopy on Oct. 6, 1945, showed the esophageal mucosa to be of a normal pink color. The cardia at times could be seen to relax but never enough to admit the scope. During withdrawal of the scope, areas of spastic constrictions were seen to come and go along the esophagus. These were so marked that they produced a tight gripping of the scope both on introduction and withdrawal, indicating actual spasm. Alvarez⁴⁶ theorized that this phenomenon is most likely due to the pathologic changes in the myenteric plexus which led to the uncoordinated irregular contraction of the esophageal musculature. These procedures revealed no evidence of organic obstruction.

Conservative treatment for achalasia using a well-balanced diet, psychotherapy, anti-spasmodics, and frequent dilatation with an Einhorn dilator was tried for three months, with only temporary and partial benefit. Occasionally the patient could keep down an entire moderately large meal. While eating she would feel the esophagus fill up until she vomited approximately one cupful of undigested food. There was accompanying pain in the region of the larynx severe enough to cause lacerination. By drinking water and forcing it down she was able to force solid foods into the stomach. She frequently became very hungry. There was no gain in weight.

She therefore entered University Hospital on Jan. 3, 1946, when a transthoracic cardioplasty was made, following a technique similar to that for Finney's pyloroplasty, using an inner tier of chromic catgut and an outer tier of interrupted Halsted mattress sutures of fine black silk. Four finger tips could be inserted easily through the new stoma. Usual reconstruction without drainage was carried out.

The esophagus was found to be greatly dilated throughout its whole length until it reached the cardiac end where it suddenly became constricted to about 1 cm. in diameter just above the crura and continued in this constricted fashion through the esophageal hiatus. There was no evidence of muscular enlargement at the cardia. There were no other local gross evidences of abnormality of the cardia. The esophageal hiatus was more than adequate in size to palpation for the passage of the esophagus. A phrenic crush was done and the esophageal hiatus was again palpated but was found not to have changed appreciably in size or shape. The disease in this one patient would appear not to have been due to a phrenospasm.

The postoperative course was smooth. She was discharged on the twenty-first postoperative day on a regular diet. Subsequently she suffered none of the former symptoms. Her general health was greatly improved. She had gradually gained weight to the present 93 pounds.

Protocol of M. J., No. 465143.—The patient, a colored man, 26 years of age, entered University Hospital on July 23, 1946. He sustained the first attack of vomiting and dyspnea ten years previously. The vomitus consisted of undigested food which frequently had a sour taste and at times a foul odor. During the three to four years before admission vomiting had become more severe and occurred at least once after each meal. Nausea seldom was associated. A feeling of fullness of the chest and palpitation often followed a meal. There had been a loss of about fifteen pounds during the three months prior to admission.



Fig. 3.—A, X-ray film studies during barium swallow on Oct. 4, 1945, showed practically no barium passing through the narrowed cardiac end of the esophagus. There was a marked fusiform dilatation of the esophagus above the cardia. B, Same case, three months postoperatively, showed definite decrease in size of the esophagus and great increase in emptying of the barium into the stomach. C, Same case, two and one-half years following a Finney type of cardioplasty. This was the nearest return of the esophagus to normal clinically and physiologically that we had observed following operation. Roentgenography on July 22, 1948, showed the esophagus to have a normal diameter. There remained no fluid level in the esophagus. In fact, this view with thick barium was difficult to obtain because of rapid and complete emptying.

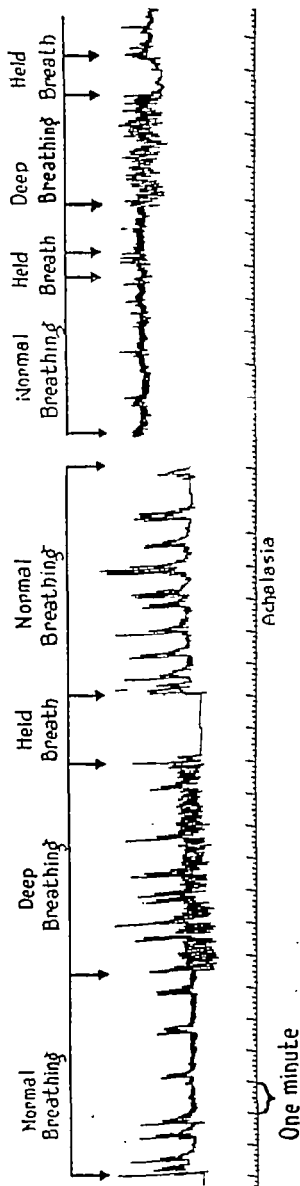


Fig. 4.—Tracing of esophageal motility of achalasia (O.P.) taken on May 5, 1918. During this period of infrequent and irregular contractions of low amplitude or deglutition was minimal. In contrast to normal, the character of the breathing played a definite part in changing the character of the tracing. When the breath was held, there was at times no evidence of intrinsic motility, and at other times the contractions were irregular and shallow.

Physical examination disclosed a cheerful, colored man. He was well developed but undernourished. The teeth were carious. The reflexes were for the most part hypoaactive.

The routine laboratory tests were negative. Radiographic study of the chest (Fig. 5, *A* and *B*) showed the typical evidences of achalasia of the esophagus. Esophagoscopy revealed a pale granular mucosa. The esophagus was tremendously dilated, tortuous, and in folds so that examination was not entirely satisfactory. No organic basis of obstruction of the cardia was found.

Conservative measures, including repeated attempts at dilatation with an Einhorn dilator were unsuccessful and uncomfortable. It was therefore felt advisable to treat this intractable achalasia by a transthoracic esophagostomy.

The usual preoperative measures were followed, including use of penicillin, sulfasuxadine, daily lavage of the esophagus, a liquid diet, and adequate additions of the usual vitamins. Under general anesthesia, using Pentothal Sodium intravenously for induction and cyclopropane, ether, helium, and oxygen intratracheally, a transthoracic cardioplasty of the Finney type was carried out, using an inner tier of catgut and two outer tiers of fine black silk. Four finger tips could be inserted through the new stoma. Penicillin was used locally. Stomach attachments, repair of the diaphragm, and closure of the chest wound were made in the usual fashion. Drainage was not used.

The pathology was merely an exaggeration of that found in O. P. At the constricted area there was no hypertrophy of the muscularis layers and no evident pathology of the mucosa. The muscularis layers as well as the mucosa of the hugely dilated portion of the esophagus were greatly thickened.

The postoperative period was entirely uneventful. Soon after discharge on the fourteenth day he began to eat a regular diet. He gained twenty-five pounds in body weight during the first year after operation.

RESULTS AND DISCUSSION

Three normal persons served as controls. They were studied under varying conditions over many hours. The character of the intrinsic motility of the normal esophagus varied greatly not only from person to person at the same level of the esophagus but at various levels and at various times at the same level in the same person. Normally, however, changes in the character of the breathing produced no appreciable change in the type of tracing (Fig. 6, *upper*) when the intrinsic motility was vigorous enough to result in good deglutition effect.

In the normal esophagus, the balloon could not be distended with over 20 to 40 c.c. of air, the amount depending upon the location of the balloon and the type of person, without producing pain. We agree with Jones⁵⁵ that sudden distention of a local segment of the esophagus was much more likely to produce a painful sensation than the more gradual introduction of air through the tube into the balloon. In our patients, also, the localization of the discomfort was usually sharply defined substernally and corresponded fairly closely with the actual level of the balloon. Most subjects complained of an uncomfortable fullness in the upper portion and more frequently of a burning sensation as the balloon was inflated at the lower levels of the esophagus. These sensations were sometimes localized in the back and rarely in the neck or face.

Normally there occurred almost a persistent intrinsic esophageal motility which was elicited by a nonpain-producing inflated balloon held in place by adequate anchorage of the tube. This balloon therefore acted as a bolus of food.

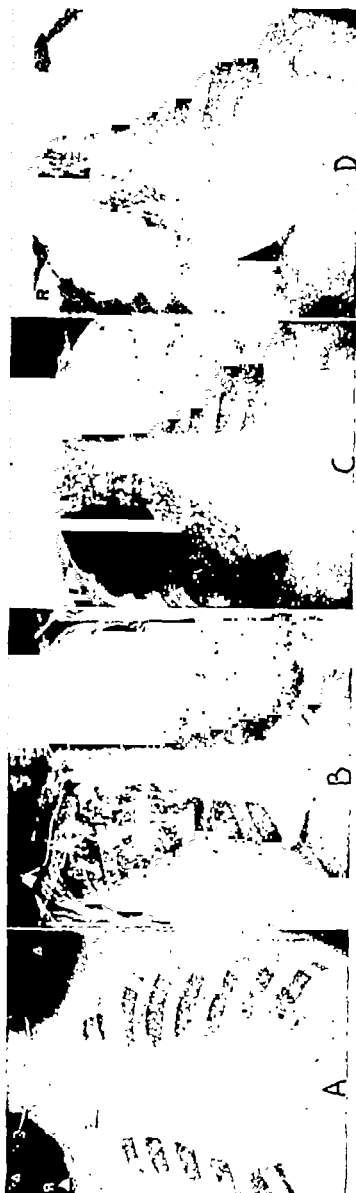


Fig. 5-1. Radiogram of chest showed a diffuse marked widening of the mediastinum. At the level of the clavicles there was a definite fluid level. B, Film studies during barium meal swallow showed a colossal air-mold mega-esophagus. Only a small amount of the ingested barium trickled into the stomach. C, Radiographic studies seventeen months following esophagostomy (Finney type) showed slight decrease in the size of the esophagus but not more than after adequate decompression of the esophagus before operation. The esophagus was still markedly dilated and an esophageal fluid level persisted in spite of a wide-open stomach. D, Same case, two and one-half years following operation showed some findings as in C.

The motility was active, responsive to stimulation, well coordinated, and effective. It produced powerful peristaltic waves and an excellent deglutition effect which was noted as a strong, persistent pull on the Einhorn tube even when the subject was placed in Trendelenburg position. This tracing (Fig. 6, *upper*) was typical of the lower esophagus (34 cm. from the incisor teeth) of R. E., a normal subject 40 years of age, as taken on Dec. 31, 1947. The balloon was slowly inflated with 30 c.c. of air, which was the maximum which could be introduced without causing pain. The shallow notches superimposed on the intrinsic contractions represent transmitted respiratory (so-called esophageal respirations) and vascular (so-called esophageal pulsations) movements.

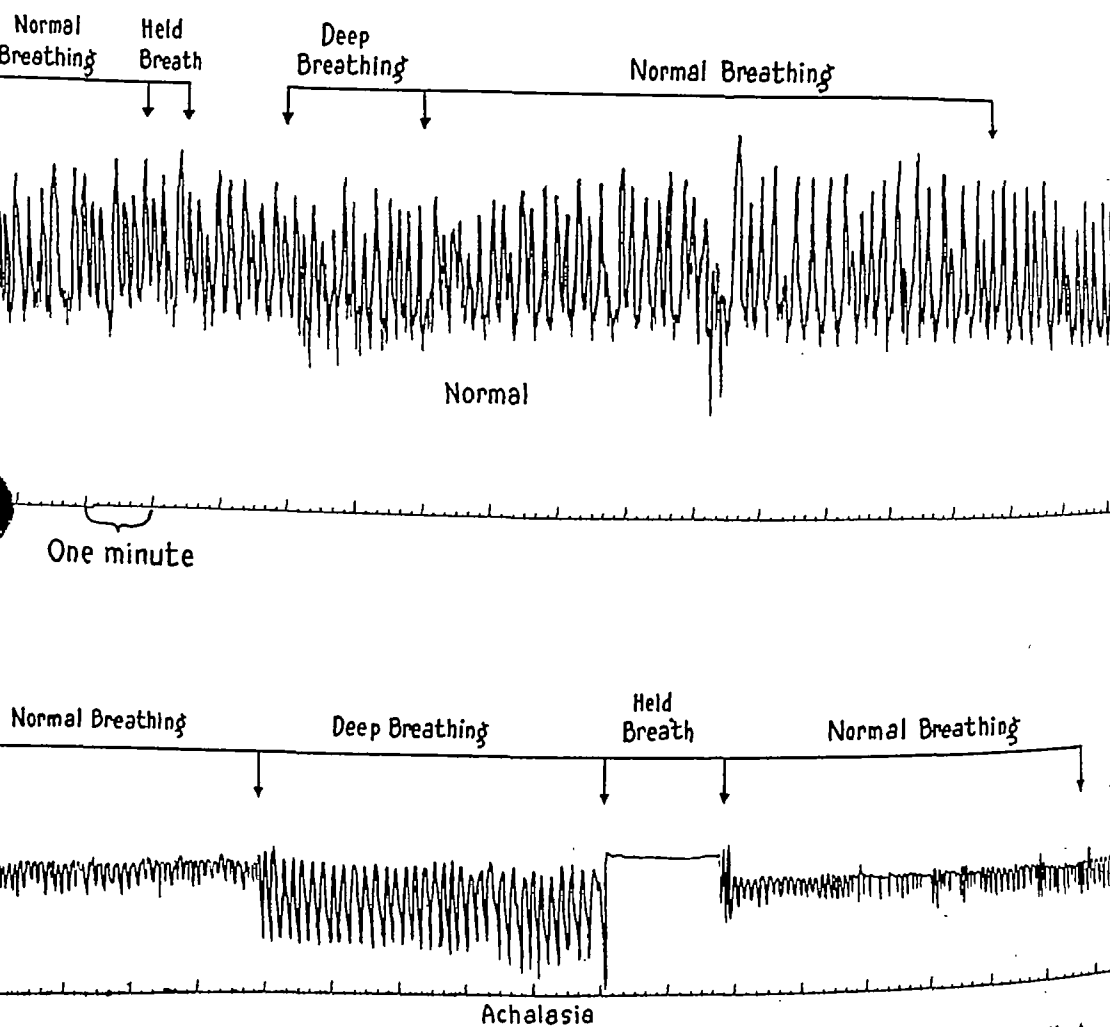


Fig. 6.—Tracings of esophageal motility of a normal person (*upper*) and of a patient with achalasia (*lower*).

The tall, regular, and frequent contractions of the normal esophagus (*upper*) produced powerful peristaltic waves and a strong deglutition effect. Changes in character of the breathing produced no appreciable change in the character of the intrinsic motility of the esophagus. In marked contrast to normal, the esophagus (M.J.) of achalasia (*lower*) showed continuous absence of intrinsic motility and no deglutition effect was produced as measured by absence of a detectable pull on the Einhorn tube. This tracing represents esophageal respirations which are synchronous with and due to the respiratory movements of the chest. When respiration was held there were no esophageal movements.

The number of contractions which occurred normally varied greatly from one every three minutes over a short duration to an average of seven every minute over long periods of time.

The amplitude of the normal contractions also varied greatly from short periods of feeble ones of less than 5 mm. of bromoform to long periods of tall ones averaging 7.5 cm. in height. The contractions of frequent occurrence and of vigorous character were accompanied by the strongest deglutition effect.

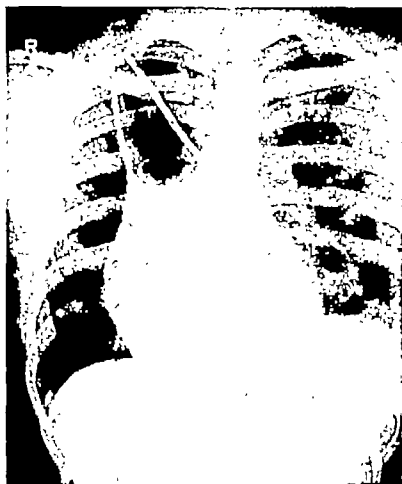


Fig. 7.—Achalasia (M.J.). When the patient was in the upright position, no matter in what portion of the esophagus we placed the balloon it always floated to the top of the esophageal fluid just as the balloon was inflated. This was in marked contrast to the normal and to O.P. No deglutition effect was produced on this balloon even when it was inflated to the point of substernal pain and well against the wall of the hugely dilated esophagus. This necessitated introduction of 500 c.c. of air.

The administration of atropine sulfate, $\frac{1}{150}$ gr. subcutaneously, to normal subjects produced in a short time a definite period of relative quiescence with a great decrease in the number and height of contractions. A decrease in the deglutition effect accompanied. Administration of urecholine, 5 mg. subcutaneously, to a normal person produced a diametrically opposite effect.

We operated on the two patients with mega-esophagus included in this study, not as an initial procedure but only after so-called conservative measures, such as psychotherapy, medical therapy, including vitamins and antispasmodics, and repeated dilatation of the cardia by a dilator had failed to give adequate relief after a reasonable trial. A type of esophagogastrostomy was employed similar to Finney's pyloroplasty and the technical end result of the operation is illustrated in Fig. 8. As explained previously, observations during operation revealed no evidence that the mega-esophagus was due to a phrenospasm.

The clinical result was good for both patients. Vomiting no longer occurred after operation; the dysphagia, if present, was minimal, and the chest pain and dyspnea disappeared. Both patients gained body weight quite rapidly and maintained a good nutritional balance. The presence of an adequate stoma in each case was demonstrated by the good clinical end results as well as by esophagoscopy observations. Yet, peristaltic deglutition as observed by use of the fluoroscope revealed this function of the esophagus much more active, coordinated, and responsive to stimulation for one patient (O.P.) so that it was difficult to obtain a good film of the esophagus (Fig. 3, C) because of the rapid emptying of thick barium on July 22, 1948, two and one-half years following operation. Similar fluoroscopic studies showed the other mega-esophagus (M. J.) was atonic, and

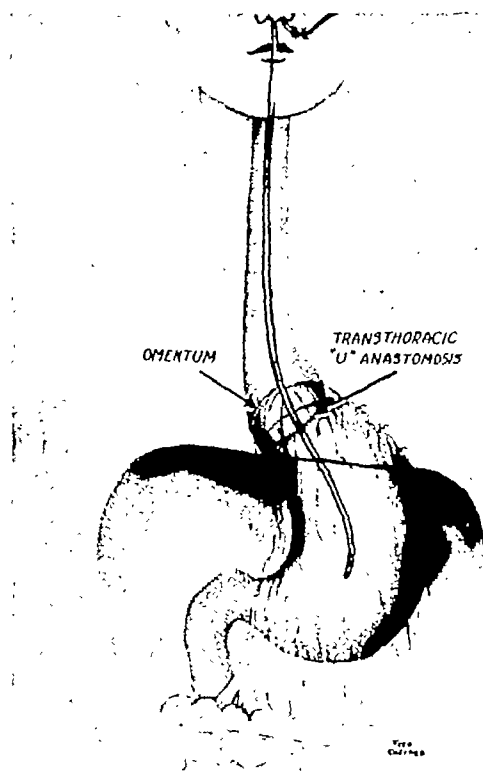


Fig. 8.—Representation of the operative end result.

unable to evacuate its contents properly, so that an x-ray film (Fig. 5, D) on Feb. 1, 1949, two and one-half years after operation, still showed the presence of an 18 cm. esophageal fluid level. Furthermore, over the same length of time postoperatively, the esophagus gradually returned to a normal size by July 22, 1948, in one patient, O. P. (Fig. 3, C), while the hugely dilated esophagus of M. J. (Fig. 5, D) did not appear to reduce in size appreciably by Feb. 1, 1949, over its decrease in size following adequate preoperative decompression. The latter phenomena have been noted by many surgeons^{43, 44} following what seemed to be an adequate cardioplasty in many cases of mega-esophagus and even after suc-

successful dilatation of the cardia. Jackson³⁴ stated that symptomatic relief is obtained by dilatation, but the associated diffuse mega-esophagus is permanent.

To our knowledge the latter phenomena have not been adequately explained. The problem of the underlying physiopathology of achalasia is further important since, we are convinced, the late postoperative functional end results on careful inquiry are frequently not as pleasing when the esophagus remains atonic and dilated. For instance, the patient (M. J.), who had the persistent mega-esophagus postoperatively, complained of inability to eat rapidly because on rapid eating there developed a sensation of nausea and of temporary holdup of food in the esophagus. The esophageal contents slowly emptied into the stomach, and it was only through slow eating that he was able to remain symptomless and to reach and maintain a normal nutrition. The other patient (O. P.), who obtained postoperative clinical, physiologic, radiologic, and esophagoscopic end results practically approaching the normal, was able to eat rapidly without symptoms. Much thought has also been given to the question as to whether mega-esophagus represents in some cases a complete motor paralysis of this structure. We have, therefore, made extensive investigations of the esophageal motility of two patients with achalasia and have compared these findings with those obtained of the normal esophagus.

We have made several motility studies, each lasting a few hours, at various times from twenty-two to twenty-six months after adequate esophagogastrostomy for O. P. The balloon was inflated with 20 c.c. of air, which was the most that this patient would tolerate without resulting in substernal pain. They showed almost continuous periods of irregular contractions of variably low amplitude and infrequent occurrence (Fig. 4). *Esophageal respirations* were prominent in this achalasia tracing (Fig. 4).

The number of contractions (O. P.), with the inflated balloon held in constant place and acting as a bolus of food, varied from one every three minutes over a short duration to four every minute over long periods, the average being one-half the normal.

The amplitude of the contractions (O. P.) also varied greatly but not as much as normal. The contractions varied from a few very shallow waves to frequent ones of an average of 2.5 cm. (bromoform) tall. Their amplitude averaged one-third the normal. As in normal persons, contractions of more frequent occurrence and of greater amplitude were accompanied by a better deglutition effect.

The administration of atropine sulfate, $\frac{1}{150}$ gr., subcutaneously, to O. P. abolished the intrinsic activity to a great extent just as for the normal esophagus. During this period the active deglutition effect was practically abolished. Administration of urecholine, 5 mg. subcutaneously, produced a diametrically opposite effect with establishment of vigorous contractions which produced so much pain on contracting over the inflated balloon that the air had to be released, and the balloon was taken into the stomach.

We made several studies of the movements of the esophagus over many hours, seventeen to twenty-nine months following adequate esophagogastrostomy for M. J. They showed complete absence of the motor function of the esophagus

(Fig. 6, *lower*) and no peristaltic activity was produced as measured by the absence of a detectable pull on the Einhorn tube carrying the inflated balloon. When the patient was in the upright position, no matter in what portion of the esophagus we placed the balloon it always floated to the top of the esophageal fluid just as the balloon was inflated (Fig. 7). This is another evidence of the absence of peristalsis of the mega-esophagus of this patient. These findings are in sharp contrast to the normal or to the findings (Fig. 1) in the other case (O. P.) of achalasia presented herewith. *Esophageal respirations*, which are synchronous with and due to the respiratory movements of the chest, are outstanding (Fig. 6, *lower*). They vary, as is readily appreciated (Fig. 6, *lower*) with the character of the respiration and are due to changes in the degree of the negative intrathoracic pressure. With increasing inspiration there occurs an increasing negative intrathoracic pressure, an increasing inflation of the balloon, and an increasing inrush of air from the manometer closed system into the balloon and a production of the lowest level of excursion on the kymogram at the point of deepest inspiration (Fig. 6, *lower*, deep breathing). Certain patients with achalasia unknowingly use these mechanics as an aid in overcoming the increased resistance at the cardia by taking a deep breath, placing the chin on the chest, and increasing the intrathoracic pressure and thereby the intraesophageal pressure by a forced expiratory effort against the closed glottis and closed cricopharyngeal sphincter which work normally in achalasia, as shown by others as well as by our cases. Contrary to these findings, it was felt heretofore that peristalsis occurred normally in the colossal mega-esophagus⁵⁴ but that it was ineffective in deglutition because the bolus of food was not large enough to be grasped by the hugely dilated esophagus.

When the patient is placed in Trendelenburg position after barium swallow, the barium rushes freely into the esophagus from the stomach in the patient in whom the function of the cardia is interfered with by resection of the esophagogastric junction as for carcinoma of the lower esophagus or by a Finney type of cardioplasty as for esophageal achalasia. This backward rush is suddenly stopped by the cricopharyngeus sphincter so that regurgitation does not occur. This condition offered an admirable opportunity to observe the almost continuous and presumably normal esophageal peristalsis in a patient 75 years of age who was symptomless two and one-half years after I had performed a radical esophagogastrectomy with a low esophagogastrostomy repair in the treatment of a squamous-cell carcinoma of the lower esophagus. These studies revealed that normally true peristalsis of the esophagus consisted of a wave of contraction passing along the esophagus at a fairly rapid speed. This wave was strong and frequently propelled the heavy barium contents uphill before it for 5 to 8 cm. from the cricopharyngeus and at times for the whole length of the esophagus. Thus it emptied completely the esophagus of its barium contents as the contraction proceeded. There occurred no detectable leakage of barium into the contracted area of the esophagus until the contraction wave came to a stop, at which time relaxation of the whole esophagus allowed the barium to rush again to the cricopharyngeus and the process was repeated. These peristaltic waves occurred more frequently and were more effective when the patient attempted to swallow

voluntarily. The peristaltic waves of the esophagus did not conform to the classic description of peristalsis as set forth by Bayliss and Starling.⁵⁷⁻⁵⁹ The wave of contraction was not preceded by a detectable wave of active relaxation and enlargement as concluded by Bayliss and Starling to receive the onflowing radiopaque barium. This conforms to the recent work of McLaren⁶⁰ who reached conclusions similar to ours after the ingenious use of roentgenkymography during barium swallow with the patient upright. Nor did suction action of the third phase or the squirting action of the pharyngeal phase of swallowing⁵⁰⁻⁵² play an appreciable role in esophageal swallowing normally when the patient was in Trendelenburg position. Normally, the propulsion of esophageal contents is therefore dependent to a great extent upon an effective peristalsis.



Fig. 9.—The bolus of thick barium (A) being swallowed by M.J. while in acute Trendelenburg position took a normal course to enter the esophageal pool (B) without spillage from the pool into the pharynx.

Further inquiry into the movements of the esophagus in achalasia in a patient (O. P.), in whom postoperatively the esophagus appeared to return to near normal clinically, radiologically, and esophagoscopically, revealed an irregularity and a great decrease below normal in the strength of the peristaltic waves. In the Trendelenburg position the barium contents rushed rapidly against the cricopharyngeus sphincter. The wave of contraction was weak and usually produced a feeble undulation of the esophagus. The barium contents were never propelled uphill before it for over 2 to 3 cm. and this occurred only when attempts were made to swallow voluntarily. The barium contents were never detectably emptied into the stomach during Trendelenburg position.

In a patient with achalasia (M. J.) who received less anatomic benefits from esophagogastrostomy than O. P., there occurred at no time discernible peristalsis. The barium meal, which collected against the cricopharyngeal sphincter during

Trendelenburg position, lay there like water in a stagnant pool. This is similar to the decreased motor activity of the stomach which is at times seen following a vagotomy.⁶² This lack of peristaltic deglutition to aid Magendie's classical third stage of swallowing indicates widespread vagal paralysis of the esophagus. It may represent an underlying primary physiopathologic mechanism; yet it could constitute a secondary alteration of the physiology of the whole muscular tube based on an advanced complicated stage of the disease. Interestingly enough, barium swallowed by M. J. while in acute Trendelenburg position took a normal course to enter the esophageal pool of barium without detectable spillage from the pool into the pharynx (Fig. 9). These motility and radiologic findings in achalasia are similar to the findings which I shall publish later⁶⁴ of two other patients with achalasia studied preoperatively.

Our patient, M. J., with achalasia sustained definite pain when a segment of the esophagus was stretched by an inflated balloon. It is reasonable to believe this another indication that the sympathetic innervation remained intact.⁶³ These data contradict the earlier consensus that pain in achalasia is conveyed by vagal afferent fibers as the problem was reviewed in 1930.⁶¹

These data suggest that we revise present views concerning the extent of the esophagus involved in achalasia. They indicate that the condition is not localized necessarily to the cardiac end of the esophagus, as is frequently stated by many authorities, but that functional abnormalities may often involve the whole length of the esophagus. These findings emphasize that our views of the extent of the cardiac operation must be better formulated as well as the postoperative treatment of the late functional condition improved in certain cases in an attempt to obtain a more normal functional end result.

SUMMARY AND CONCLUSIONS

1. Three normal persons served as controls. They were studied under varying conditions over many hours. These studies show that organized frequent contractions of good amplitude are related normally with effective active peristalsis of the esophagus and play an outstanding role in Magendie's third stage of swallowing.

2. For two cases of intractable achalasia of the esophagus, a type of trans-thoracic esophagogastronomy was employed similar to Finney's pyloroplasty. Observations during operation revealed no evidence that the mega-esophagus was due to a phrenospasm.

3. One of the patients with achalasia showed the nearest return of the esophagus to normal clinically, physiologically, radiologically, and esophagoscopically that we have observed following esophagogastronomy. Motility studies disclosed almost continuous periods of irregular contractions of infrequent occurrence and variably low amplitude. The contractions were not well organized, so that minimal strength of peristalsis was produced as measured by minimal pull on the Einhorn tube carrying a balloon inflated lightly against the wall of the esophagus. However, the contraction waves had sufficient power to force the balloon into the stomach and the deglutition effect was strong enough to produce

very good emptying of the esophagus through the large operative stoma when the patient was in the upright position. On the other hand, emptying of the esophagus was not appreciable in the Trendelenburg position. As in normal persons, contractions of more frequent occurrence and of greater amplitude were accompanied by a better deglutition effect.

4. Another patient with achalasia gained about as much amelioration of symptoms following esophagogastrostomy and yet the hugely dilated esophagus decreased but slightly in size over a period of two and one-half years following operation, a condition which has been noted by other surgeons. Radiologically, the esophagus was unable to evacuate properly its contents so that an esophageal fluid level persisted. Motility studies showed continual absence of the motor function of the esophagus, and no peristaltic activity was produced as measured by the absence of a detectable pull on the Einhorn tube.

5. Patients whose function of the cardia was abolished by operative procedures offered an admirable opportunity to observe the movements of the esophagus for the reason that when placed in Trendelenburg position after barium swallow the barium rushed freely into the esophagus from the stomach to be stopped suddenly by the cricopharyngeal sphincter so that regurgitation did not occur. The normal movements of the esophagus of a patient thus studied two and one-half years following excision of a carcinoma of the lower esophagus frequently emptied the esophagus of its barium but did not conform to the classic description of peristalsis, since the wave of contraction was not preceded by a wave of active relaxation. In achalasia the movements were uncoordinated or absent and the strength of the contraction wave was greatly decreased below normal or absent, so that the esophagus in Trendelenburg position was never appreciably emptied of its barium.

6. Information obtained by barium swallow and by observed clinical effects on the inflated balloon which acted as a bolus of food was complementary to that obtained from the esophageal motility studies. The data considered together gave adequate conceptions of the action of the musculature of the esophagus. These data indicated that the intrinsic motility of mega-esophagus varies greatly, being often irregular, uncoordinated, hyporesponsive to stimuli, and not completely effective. In fact the intrinsic motility may be entirely absent and completely fail. This differs from that of the organic obstructive lesions of the esophagus in which there occurs usually a normal motility or a hypermotility but rarely even a hypomotility, depending upon the degree of obstruction and the stage and complications of the organic disease. Achalasia of the esophagus also differs from true esophagospasm in which there may occur a hypermotility.

7. A disturbed peristalsis or even total motor paralysis of the whole esophagus may be present in this disease. The latter is the underlying factor which explains the inability of the esophagus to return to normal size following an adequate esophagogastrostomy. It may also explain some of the failures of this operation. Our studies indicate that the stoma must be made sufficiently large and constructed in such a manner as to offer the least resistance to gravitational outflow of the esophageal contents into the stomach since the factor of peristaltic force is here diminished or even abolished.

8. Our data indicate that the disease is not localized necessarily to the cardiac end of the esophagus as is commonly believed, but that functional abnormalities may often involve the entire length of the esophagus.

9. It seems irrational to use a member of the atropine series in the treatment of true achalasia since atropine sulfate produced a further depressant effect on the esophageal motility and peristalsis. Atropine, however, does decrease the amount of salivation which may result in symptomatic relief. Our data indicate that a sympathicolytic drug such as ergotamine or Priscol and a drug which has the peripheral effects of acetylcholine, such as urecholine, are worthy of a reasonable clinical trial in the conservative treatment of achalasia and in certain cases in the adequate postoperative treatment of the patient.

10. Normal accuracy of localization of the inflated balloon is generally present in achalasia so that the sensory supply presumably remains undisturbed. Recent data contradict the earlier consensus that pain in achalasia is conveyed by vagal afferent fibers.

11. These newer observations offer further evidence for and are compatible with the general theory of achalasia as an underlying mechanism of cause of idiopathic mega-esophagus.

REFERENCES

1. Hurst, Arthur F.: Case of Achalasia of the Cardia (So-called Cardiospasm), *Proc. Roy. Soc. Med. (Clin. Sect.)* 8: 22, 1914.
2. Hurst, A. F., and Rake, G. W.: Achalasia of the Cardia (So-called Cardiospasm), *Quart. J. Med.* 23: 491, 1930.
3. Willis, T.: *Pharmaceutice Rationalis*, First English Translation, London, 1679, p. 23.
4. Einhorn, M.: A Case of Dysphagia With Dilatation of the Esophagus, *Med. Rec.* 34: 751, 1888.
5. Rolleston, H. D.: Simple Dilatation of the Esophagus, *Tr. Path. Soc. Lond.* 47: 37, 1896.
6. Vinson, P. P.: *The Diagnosis and Treatment of Diseases of the Esophagus*, Springfield, Ill., 1940, Charles C Thomas, Publisher, p. 86.
7. Boyd, W.: *Surgical Pathology*, Philadelphia, 1943, W. B. Saunders Company, p. 169.
8. Wright, Samson: *Applied Physiology*, London, 1945, Oxford University Press, p. 759.
9. Kronecker, Hugo, and Meltzer, Samuel: *Arch. f. Anat. u. Physiol. (Supp.)* p. 328, 1883.
10. Rake, G. W.: On the Pathology of Achalasia of the Cardia, *Guy's Hosp. Rep.* 7: 141, 1927.
11. Cameron, J. A. M.: Oesophagectasia in a Child, *Arch. Dis. Childhood* 2: 358, 1927.
12. Beattie, W. J. H. M.: Achalasia of the Cardia, *St. Barth. Hosp. Rep.* 64: 39, 1931.
13. Etzel, E.: Os nervos pneumogástricos no megasôfago. Estudo anatomopatológica de 5 casos, *Ann. Fac. de med. da Univ. de S. Paulo* 10: 373, 1934.
14. Lendrum, F. C.: Anatomic Features of the Cardiac Orifice of the Stomach With Special Reference to Cardiospasm, *Arch. Int. Med* 59: 474, 1937.
15. Tamiya, C., and Tamura, M.: Röntgenologisch-experimentelle Studien über den Oesophagus mit besonderer Rücksicht auf die Pathogenese der sogenannten "funktionellen" idiopathischen Oesophagusdilatation, *Ztschr. f. d. ges. exper. Med.* 78: 337, 1931.
16. Etzel, E.: A avitaminose como agente etiologico do megasôfago e do megacolo, *Ann. Fac. de med. da Univ. de S. Paulo* 11: 59, 1935.
17. Knight, G. C.: The Relation of the Extrinsic Nerves to the Functional Activity of the Esophagus, *Brit. J. Surg.* 22: 155, 1934.
18. Ferguson, J. H.: Effects of Vagotomy on the Gastric Function of Monkeys, *Surg., Gynec. & Obst.* 62: 689, 1936.
19. Ozorio de Almeida, M.: Sur la dilatation chronique de l'oesophage produite expérimentalement chez le chien par section partielle des pneumogastriques, *Compt. rend. Soc. de biol.* 102: 407, 1929.
20. Knight, G. C.: Sympathectomy in Treatment of Achalasia of Cardia, *Brit. J. Surg.* 22: 864, 1935.
21. Stubbe, H.: Oesophageal Achalasia Treated by Sympathectomy, *M. J. Australia* 24: 1001, 1937.

22. Souttar, H. S.: Oesophageal Obstruction, Brit. M. J. 2: 777, 1935.
23. Craig, W. M., Moersch, H. J., and Vinson, P. P.: Treatment of Intractable Cardiospasm by Bilateral Cervicothoracic Sympathetic Ganglionectomy, Report of a Case, Proc. Staff Meet., Mayo Clin. 9: 749, 1934.
24. Turner, G. G., and Knight, G. C.: Surgery of the Oesophagus, Tr. M. Soc., London 59: 171, 1936.
25. Woollard, H. H.: The Peripheral Sympathetic Nervous System, Brit. J. Surg. 23: 425, 1935.
26. Mitchell, G. A. G.: The Nerve Supply of the Gastro-Oesophageal Junction, Brit. J. Surg. 26: 333, 1938.
27. von Mikulicz, J.: Ueber Gastroskopie und Oesophagoskopie, Mitth. d. Ver. d. Aerzte in Nied. Oesterreich 8: 41, 1882.
28. Huss, M.: Dilatatio oesophagi ingluviformis, Hygien 4: 290, 1842.
29. Carlson, A. J., Boyd, T. E., and Penney, J. F.: The Innervation of the Cardia and the Lower End of the Esophagus in Mammals, Ann. J. Physiol. 61: 14, 1922.
30. Openchowski, T.: Ueber die nervösen Vorrichtungen des Magens: Experimentelle Studien, Zentralbl. f. Physiol. 3: 1, 1889.
- Idem: Ueber die gesammte Innervation des Magens, Deutsche med. Wchnschr. 15: 717, 1889.
- Idem: Zur pathologischen Anatomie der geschwürigen Prozesse im Magendarmtractus, Arch. f. path. Anat. u. Physiol. 117: 347, 1889.
31. Meyer, W.: Impermeable Cardiospasm Successfully Treated by Thoracotomy and Oesophagoplication, Ann. Surg. 53: 293, 1911.
32. Sauerbruch, F.: Der Kardiospasmus, in Chirurgie der Brustorgane, Vol. II, Berlin, 1925, Julius Springer, p. 572.
33. Rieder, W.: Klinik und Therapie des sogenannten Cardiospasmus, Deutsche Ztschr. f. Chir. 222: 47, 1930.
34. Pieri, G.: Contributi clinici alla chirurgia del sistema nervoso vegetativo; la cura della stenosi esofagea (cardiospasm), Arch. ital. di chir. 35: 644, 1933.
35. Sauerbruch, F., and von Hacker, R.: Zur Frage des Cardiaverschlusses der Speiseröhre, Deutsche med. Wchnschr. 32: 1263, 1906.
36. Jackson, C.: The Diaphragmatic Pinchcock in So-called "Cardiospasm," Laryngoscope 32: 136, 1922.
37. Hill, W.: Dilatation of Esophagus Without Anatomical Stenosis, Proc. Roy. Soc. Med. 12: 33, 1919.
38. Womack, N. A.: Esophagoplasty for Esophageal Achalasia, S. Clin. North America 18: 1241, 1938.
39. Ballou, H. C., Wilson, H. M., Singer, J. J., and Graham, E. A.: Esophagus, Stomach and Heart Following Unilateral Phrenicectomy, Arch. Surg. 21: 1291, 1930.
40. Turner, G. G.: Some Experiences in Surgery of the Esophagus, New England J. Med. 205: 657, 1931.
41. Vampré, E.: Le "mal de engasgo" et son traitement chirurgical, Rev. sud-am. de méd. et de chir. 4: 493, 1933.
42. Fromme, A.: Erfahrungen mit der Operation des Kardiospasmus (see Discussion), Arch. f. klin. Chir. 157: 606, 1929.
43. Ochsner, Alton, and De Bakey, Michael: Surgical Considerations of Achalasia; Review of the Literature and Report of Three Cases, Arch. Surg. 41: 1140, 1940.
44. Clagett, O. Theron, Moersch, Herman J., and Fischer, Albert: Esophagogastrostomy in the Treatment of Cardiospasm, Surg., Gynec. & Obst. 81: 440, 1945.
45. Puppel, I., Darin, Brown, Ray, Lacey, Paul, and Cox, Lester: Instruments Useful in Esophagogastrintestinal Motility Studies. Unpublished data.
46. Alvarez, Walter C.: An Introduction to Gastroenterology, New York, 1940, Paul B. Hoeber, Inc., p. 197.
47. Bignami, G.: Megnesofago e megndolicocolon, Riv. di radiol. e fis. med. 6: 671, 1931.
48. Ledoux-Lebard, R., Nemours-Auguste, R., and Lefebvre, J.: Un cas de dilatation idiopathique de l'oesophage associée a un dolicho-côlon, Arch. d. mal. de l'app. digestif 26: 1085, 1936.
49. Martini, T., and Comas, J.: Formas esofágicas de la enfermedad de Glennard-Lane, Semana méd. 1: 1320, 1937.
50. Barclay, A. E.: Normal Mechanism of Swallowing, Brit. J. Radiol. 3: 534, 1930.
51. Barclay, A. E.: The Normal Mechanism of Swallowing, Proc. Staff Meet., Mayo Clin. 5: 251, 1930.
52. Barclay, A. E.: Der normale Schlingmechanismus, Acta. radiol. 13: 87, 1932.
53. Barclay, A. E.: The Digestive Tract: A Radiological Study of Its Anatomy, Physiology, and Pathology, London, 1933, Cambridge University Press.
54. Negus, V. E.: The Mechanism of Swallowing, Proc. Roy. Soc. Med. 36: 85, 1942.
55. Jones, C. M.: Digestive Tract Pain, New York, 1938, The Macmillan Company, p. 7.

56. Jackson, C., and Jackson, C. L.: *Diseases of the Nose, Throat and Ear*, Philadelphia, 1945, W. B. Saunders Company, p. 729.
57. Bayliss, W. M., and Starling, E. H.: *The Movements and Innervation of the Small Intestine*, *J. Physiol.* 24: 99, 1899.
58. Bayliss, W. M., and Starling, E. H.: *The Movements and Innervation of the Large Intestine*, *J. Physiol.* 26: 107, 1900.
59. Bayliss, W. M., and Starling, E. H.: *The Movements and Innervation of the Small Intestine*, *J. Physiol.* 26: 125, 1901.
60. McLaren, J. W.: *Kymography and Its Application to Esophageal Movement*, *Brit. J. Radiol.* 16: 270, 1943.
61. Patten, C. A.: *The Vegetative Nervous System: An Investigation of the Most Recent Advances*, Baltimore, 1930, Williams and Wilkins Company, p. 832.
62. Barron, L. E., and Curtis, G. M.: *Effect of Vagotomy on the Gastric Motor Mechanism*, *Arch. Surg.* 34: 1132, 1937.
63. White, J. C., and Smithwick, R. H.: *The Autonomic Nervous System: Anatomy, Physiology and Surgical Application*, New York, 1941, The Macmillan Company, p. 142.
64. Puppel, I. Darin: *The Pathologic Physiology of Mega-esophagus*, *J. Lab. & Clin. Med.* 34: 1739, 1949.

PERMANENT FILLING OF DEAD SPACE IN THE PLEURAL CAVITY AFTER PNEUMONECTOMY

AN EXPERIMENTAL STUDY

JOHN H. GRINDLAY, M.D.,* O. THERON CLAGETT, M.D.,† AND
JOHN R. RYDELL, M.D.,‡ ROCHESTER, MINN.

THE purpose of this experimental study was to test whether a lightweight, plastic prosthesis, roughly the size and shape of the lung, could be used to occupy the dead space left in the pleural cavity after removal of one lung. A prosthesis was placed in the pleural cavity after right or left pneumonectomy in a series of twenty-one dogs. Postoperative studies included roentgenograms of the thorax and, in a few experiments, exploratory thoracotomy. Several dogs were killed while they were under anesthesia in order that reaction to the prosthesis might be investigated in detail. The first dogs in which the prosthesis was used have now gone nine months since operation. Because we were unable to study pleural reaction and exudate as thoroughly as we wished in dogs, especially during the early postoperative period, a similar series of experiments was carried out in a series of rats. This report, however, deals with the results of our study on dogs. A report of the study on rats will be made later by one of us (J. R. R.), and only the pertinent conclusions of the experiments on rats will be included in this report. In the sense that the experiments on dogs must be continued for a much longer period and that the prosthesis is being modified and improved, the present report is preliminary.

It was not within the scope of our study to investigate physiologic function of the remaining lung after pneumonectomy, or to compare its function when no prosthesis was used with function when a prosthesis was used in the empty pleural cavity. The possibility that respiratory function is impaired is suggested by the fact that permanent anatomic changes occur in the thorax after the removal of one lung and a segment of the phrenic nerve on that side. Narrowing of the intercostal spaces and rise of the diaphragm constrict the pleural cavity to a significant extent after the lung has been removed, the residual space is usually filled by the heart and other mediastinal structures, and the remaining lung usually enlarges to a degree necessary to compensate for the mediastinal shift. This enlargement of the lung has been called "compensatory emphysema," although it has been shown by Rienhoff, Reichert, and Heuer¹ and by Behrend and Mann² that this is not true emphysema in dogs. They found that increases in size and weight of the lung were the result of the fact that the lung was more fully expanded and contained more blood than normally. The possibility remains, however, that simple distention of the lung may eventually lead to emphy-

Read at the Forum on Fundamental Surgical Problems at the meeting of the American College of Surgeons, Los Angeles, Calif., Oct. 18, 1948.

Received for publication Dec. 8, 1948.

*Division of Experimental Medicine, Mayo Foundation.

†Division of Surgery, Mayo Clinic.

‡Fellow in Surgery, Mayo Foundation.

sematous changes in some persons. Evidence concerning the function of the lung, whether it be simply distended or pathologically distended, has been accumulated in recent years.

Cournand and Berry³ as well as Birath and co-workers⁴ have concluded that overdistention of the remaining lung after pneumonectomy reduces the breathing reserve, produces an increased tendency to dyspnea, and allows marked reduction in the oxygen saturation of arterial blood to occur during strenuous exertion. Such changes were somewhat less likely to occur if pneumonectomy was performed during childhood or early adolescence and more likely to occur in older adults than in young adults.^{3, 5} Thoracoplasty as a second operation reduced the degree of overdistention in adult patients and improved ventilatory efficiency. Similarly, prevention of overdistention in a patient who already had emphysema before pneumonectomy, by placing air or oil in the thorax on the side of pneumonectomy, caused improvement in pulmonary function.

Another method of prevention of overdistention of the remaining lung was reported on recently by Johnson and co-workers.⁶ In a preliminary report of studies on dogs they described the use of a prosthesis to occupy the empty pleural cavity after pneumonectomy. In one series of dogs observed for as long as four months, an airtight hollow box of zirconium measuring 10 by 6 by 3.5 cm. was used. In another series, hollow balls of methyl methacrylate 1 inch (2.5 cm.) in diameter, similar to those used by Wilson⁷ for an extrapleural pack, were used to fill the pleural cavity. Dogs of the latter group were observed as long as two months. A significant degree of pleural reaction was observed in dogs with the zirconium prosthesis, but only minimal pleural reaction occurred when the plastic balls were used.

Our study was the outgrowth of one in which we used gelatin foam sponges to occupy the dead space after pneumonectomy in dogs and rats.⁸ Although this material was gradually absorbed, postoperative shift of the mediastinum and gross pleural effusion were prevented. It was noted also that dogs so treated were much less dyspneic and considerably more active after operation than dogs in which gelatin foam sponge was not used. These observations suggested that function of the remaining lung was not impaired but improved, during the postoperative period, by a foreign material placed in the empty pleural cavity and encouraged us to investigate the possibilities of use of a nonabsorbable prosthesis.

We believed the most suitable prosthesis would be one which would fit the pleural cavity and leave a minimum of dead space, would be just large enough to prevent mediastinal shift, and would not produce harmful pleural reaction or other ill effects. Our desire to have the prosthesis leave as little dead space as possible and to avoid having empty spaces around the prosthesis, such as are present between balls, was based on theoretic objections that dead space would permit motion, friction, mechanical trauma, and discomfort, and that the greater the dead space the greater the volume of free fluid and the greater the danger from infection.

METHODS

At the time the study was undertaken we^{9, 10, 11} had had two years of experience with Polythene (polyethylene) in various living tissues and were impressed that it caused

neither inflammatory nor foreign body reaction in many tissues and that it was not altered by exposure to living tissue. Accordingly, it seemed that Polythene might be suitable for the thoracic prosthesis. We constructed a roughly lung-shaped bag with Polythene film (thickness 0.005 inch) by fusing the edges together with heat and pressure (Fig. 1). The bags contained air and, in order to give them the desired shape, rather tightly packed fiber glass. In flat view the cephalic end of the bag was rounded but narrow, while the caudal end was wide but sloping, so that the posteroinferior portion of the bag in situ was lower than the anteroinferior portion. The bags were slightly thicker at the base than at the apex. The density of the prosthesis was appreciably less than that of inflated lung. After the bags had been tested for leaks they were sterilized by immersion in 1:1,000 aqueous solution of benzalkonium chloride (Zephiran chloride). The inside of the bag and its contents were not sterilized.



Fig. 1.—The Polythene bag prosthesis. The outer layer of the prosthesis is Polythene film (thickness 0.005 inch), the edges of which have been sealed together by heat. The prosthesis contains air and rather tightly packed fiber glass.

The prosthesis was placed in the pleural cavity of the dogs, after right or left pneumonectomy, before the thorax was closed. A sufficient number of sterile prostheses of various sizes was always available so that one of suitable length, width, and thickness was on hand. No attempt was made to aspirate the remaining air after the thorax was closed, and thoracentesis was not performed during the postoperative period. In judging the size of the prosthesis to be placed in the thorax we allowed for the rise of the hemidiaphragm and narrowing of the intercostal spaces and tried to select a Polythene bag which would be the size of the pleural cavity during maximal expiration after such changes had taken place. Our early experience with Gelfoam taught us the danger of filling the pleural cavity to such an extent that function of the remaining lung was compromised.

RESULT OF EXPERIMENTS

Right pneumonectomy was performed on 10 dogs and left pneumonectomy on 11 dogs; each of these dogs had a Polythene bag prosthesis placed in the pleural cavity after removal of the lung. All operations were performed with the dog under ether anesthesia and with aseptic surgical technique. No difference was observed between dogs in which the right lung was removed and those in which the left lung was removed. All dogs tolerated the operation well and were in excellent condition during the early postoperative period.

Six of the 21 dogs of the series died between four and fourteen days (average seven and one-half days) after operation. Necropsy revealed that in each instance the bag had split open at a seam and that acute empyema was present. Another dog died twenty days after operation because of fibrosis around the inferior vena cava near the right atrium. A loosely attached thrombus occluded the entire thoracic part of the inferior vena cava, the liver was greatly enlarged and nearly black in color, massive abdominal ascites was present, and both pleural cavities contained massive amounts of blood stained fluid. The cause of the fibrosis about the vena cava and thrombosis could not be determined with certainty, but we believe it was mechanical trauma produced by the prosthesis. The bag was too small for this dog and could have rotated enough so that during the respiratory cycle the seam along the posterior border scratched back and forth on the cephalic end of the inferior vena cava. Another dog died on the twenty-eighth postoperative day because of erosion of the left internal mammary vein and the massive hemorrhage which followed. There seemed to be no question that in this case the seam along the apex of the bag had impinged on the vein and finally lacerated it. Two other dogs of the series died, 1 at five months and the other at six months postoperatively; the cause of death in each instance was acute empyema caused by a leak in the bag and exposure of its nonsterile contents to the pleural cavity. The leak in one bag consisted of a burst seam, in the other of a crack at the site of a wrinkle.

Two of the 11 dogs that lived were killed, while they were under ether anesthesia, in order that pleural reaction could be studied, 1 dog four and one-half months and the other seven months after operation. Both dogs appeared to be in excellent general condition and weighed more than they weighed before operation. The heart and mediastinum were in the normal position in the midthorax, and the remaining lung in each dog appeared entirely normal, both grossly and histologically. The right pleura (side of pneumonectomy) in the dog killed at four and one-half months was almost everywhere of normal thickness and appearance (Fig. 2). Slight thickening was noticed in a few areas of the parietal pleura, the principal area being over the healed operative wound. This side of the thorax contained 30 c.c. of clear straw-colored fluid. There were a few erythrocytes in the fluid, and the leucocytes numbered 2,890 per cubic millimeter, of which 5 per cent were lymphocytes and 95 per cent were neutrophils. The Polythene bag prosthesis was not completely intact. The fiber glass at the apex of the bag was stained green and was slightly moist, apparently the result of a small leak that allowed some fluid to enter the nonsterile interior of the prosthesis.

The pleural surface on the left, the side of pneumonectomy, in the dog killed at seven months likewise showed few changes. The mid-portion of the pericardial pleura, however, was pink and granular, and there were two similar pink, granular areas in the mid-parietal region, each about 3 cm. in diameter. The pleura in the region of the thoracotomy wound was opaque and gray but glistening. The remainder of the pleural surface appeared normal, and this side of the thorax contained 20 c.c. of clear, straw-colored fluid. A tiny hole was present in the corner of a wrinkle near the lower end of the prosthesis, and the air that had been in the bag was completely replaced by clear, straw-colored fluid. Although the inside of the prosthesis had not been sterilized when it was made, significant bacterial growth obviously had not occurred.

Nine dogs of the series are living and all appear to be in excellent general condition. Two have had the prosthesis in place for nine months, 2 for eight months, 2 for seven months, 2 for six months, and 1 for five months. Five of these dogs had right pneu-

monectomy and 4 left pneumonectomy. Exploratory thoracotomy was performed in 1 dog at two months and in another at three months; biopsy of the mediastinal and parietal pleurae revealed slight thickening. The gross appearance of the pleura in each dog was normal. In 1 dog 20 c.c. of free fluid was found in the pleural cavity and in the other not enough fluid was present for aspiration. The fluid in the 1 dog was pink, probably because of contamination by fresh blood.

Roentgenographic examination on the day after operation was made in 5 dogs. Except for slight narrowing of intercostal spaces, slight rise of the hemidiaphragm, and absence of the usual pulmonary markings, the roentgenogram was that of a normal thorax. A detectable amount of fluid was not present, and the mediastinum was in the normal position. Roentgenograms of the thorax were obtained on all the dogs at about two or four weeks and thereafter at intervals of one or two months. In general, the examination



Fig. 2.—Dog killed four and one-half months after right pneumonectomy and implantation of a Polythene bag prosthesis in the right pleural cavity. The mediastinum is in normal position and the right pleural surface is slightly thickened. The lower medial portion of the right pleural cavity shows the 20 c.c. of fluid which was present at the time of necropsy.

disclosed no further change for the first four to five months. After this time the prosthesis, instead of being invisible roentgenographically, had a mottled appearance. Later, some of the prostheses became radiopaque. For a time, this finding confused us. We suspected at first that a capsule might have formed about the prosthesis and then became calcified. The dogs which were killed gave us the explanation of this finding. The roentgenogram, made just before death, of the dog killed at four and one-half months, showed mottling at the upper end of the bag; the fiber glass at this end of the bag was found to be moist. The roentgenograms of the dog killed at seven months had shown mottling of the bag at five months, and at the time of his death the bag was radiopaque. Necropsy showed that the bag was filled with fluid. We have concluded, therefore, that radiopacity of the prosthesis indicates fluid which has entered through a leak.

RECENT EXPERIMENTS

Because we learned from the series of 21 dogs that the Polythene bag prosthesis had structural weakness, that is, a sharp seam along its edge and a tendency to break open, we have discarded this prosthesis. We are continuing our study* and are now employing a hollow, air-filled, methyl methacrylate (Lucite, Plexiglass) prosthesis. The prosthesis has a perfectly smooth external surface, is very light in weight, and is molded to fit the pleural cavity. Two dogs have had this prosthesis in the left pleural cavity for two and one-half months, another for two months, and still another for one month. No deaths or complications have occurred thus far, and the mediastinum appears, on roentgenographic examination, to be in the normal position.

COMMENT

Our study has demonstrated that a prosthesis roughly the size and shape of the space remaining in the pleural cavity after right or left pneumonectomy will prevent the occurrence of mediastinal shift and, presumably, overdistention of the remaining lung. We are aware, however, that our study has been merely a test of whether a prosthesis would prevent mediastinal shift and that physiologic studies must be made to determine whether the prosthesis is effective in protection against the harmful effects of overdistention of the remaining lung. Whether our single-unit prosthesis has advantages over the multiple hollow-ball prosthesis described by Johnson⁶ remains to be determined. We question, however, that it would be more difficult surgically, as Johnson feared, to remove a large single-unit prosthesis, should the need arise, than a number of small balls. If the balls become fixed in position some of them might be hard both to find and to remove. We feel that the single-unit prosthesis, shaped to fit the pleural space, might have certain advantages over the multiple-ball prosthesis. For example, the patient might have fewer subjective symptoms because there would be no other foreign body on which the prosthesis could impinge. Also, it seems to us an advantage to provide the pleural cavity with a preformed prosthesis and a theoretic disadvantage to let uncontrollable forces collect and stabilize a number of small objects into an effective prosthesis. We suspect, furthermore, that there are dangers inherent in the small-ball prosthesis; namely, that a ball might insinuate itself into a mediastinal pocket and produce pressure on the esophagus or a large blood vessel, or that the organic material (fluid or tissue) which fills the spaces between balls, especially spaces most distant from a source of blood supply, might be pre- to infection. Finally, there may be danger of overfilling the pleural cavity, left applying pressure to the mediastinum if balls are used because the surgeon may be tempted to fill the pleural cavity completely and not to follow directions based on preoperative measurements.

The Polythene bag prosthesis, like the multiple, hollow, methyl methacrylate ball prosthesis of Johnson,⁶ produced minimal pleural reaction as long as it did not leak and allow pleural fluid to come in contact with the nonsterile contents of the prosthesis. Our observations of pleural reaction, however, are based on only 4 dogs, of which 2 were explored before the prosthesis had begun to leak, 1 dog was killed when a leak had started, and 1 was killed when the prosthesis was full of fluid. In order to obtain more information concerning pleural reac-

*In collaboration with Dr. A. H. Bulbulian.

tion than we could on a series of dogs we made a study of pleural reaction in rats. Careful determination of volume and cellular content of pleural fluid and histologic examination of the pleura were made in a large series of rats killed at frequent intervals ranging from one day to several months after pneumonectomy and implantation of a Polythene bag prosthesis. These studies showed that the prosthesis did not cause formation of excessive or abnormal pleural fluid and that pleural reaction was limited to the formation of a very thin fibrous capsule about the prosthesis. In our dogs, such a capsule has not been observed.

We recognize the structural defects of the prosthesis we have used in this study but we are confident the defects can be corrected. The molded, hollow, methyl methacrylate prosthesis that we have used lately appears to be structurally stronger than the Polythene prosthesis, although it is much more rigid; it is superior also in that it has no protruding seams. We believe that the best permanent prosthesis will be one having the general shape of the space it is to occupy, will be of just the right size to prevent appreciable mediastinal shift, will have permanent structural strength, will be as nonirritating as the prosthesis we have used, and will have so smooth a surface that there will be no danger of mechanical trauma to neighboring living tissue.

SUMMARY

A lightweight bag, made by heat-sealing Polythene film and filled with fiber glass and air, roughly the shape of the pleural cavity, and of sufficient size to prevent mediastinal shift, was placed in the pleural cavity after right or left pneumonectomy in a series of twenty-one dogs. Only the outside of the prosthesis was sterile. During the early postoperative period all dogs were in excellent condition.

Six dogs died during the first two weeks because of acute empyema caused by rupture of the prosthesis. Two dogs died, five and six months after operation, respectively, from the same cause. Two dogs died from lesions attributed to mechanical trauma of blood vessels adjacent to the prosthesis.

Two dogs were killed, one at four and one-half months and the other at seven months. The mediastinum of each was in normal position, and the pleura showed a minimal degree of thickening.

Nine dogs are well and have had the prosthesis in place for periods ranging from nine to five months. The position of the mediastinum has always been normal in these dogs.

Eventually a leak consisting of either a crack or a rupture of the Polythene covering developed in all the Polythene bag prostheses. This has permitted the air within the prosthesis to be partially or completely replaced by fluid. Although sterilizing the inside of the prosthesis and its contents would probably lessen the danger of bacterial growth in the prosthesis and of empyema, this weakness of the Polythene bag makes further use of it as a prosthesis undesirable unless the defects are corrected. Recent experience with a lightweight,

hollow, molded one-piece prosthesis of methyl methacrylate in a series of four dogs indicates that this prosthesis is superior to the one made with Polythene and filled with air and fiber glass.

This study has demonstrated that the pleural cavity tolerates a large, non-irritating prosthesis after removal of the lung and that mediastinal shift can be prevented by such a prosthesis. This study and a similar one in rats have demonstrated that the prosthesis does not cause formation of excessive amounts of fluid and that it causes minimal pleural reaction.

REFERENCES

1. Rienhoff, W. F., Jr., Reichert, F. L., and Heuer, G. J.: Compensatory Changes in the Remaining Lung Following Total Pneumectomy; an Experimental Study, *Bull. Johns Hopkins Hosp.* 57: 373-383, 1935.
2. Behrend, Albert, and Mann, F. C.: Some Postoperative Effects of Pneumectomy; a Morphologic Study, *J. THORACIC SURG.* 6: 685-697, 1937.
3. Cournand, André, and Berry, F. B.: The Effect of Pneumectomy Upon Cardiopulmonary Function in Adult Patients, *Ann. Surg.* 116: 532-550, 1942.
4. Birath, G., Crafoord, C., and Rudstrom, P.: Pulmonary Function After Pneumectomy and Lobectomy, *J. THORACIC SURG.* 16: 492-511, 1947.
5. Cournand, André, Himmelstein, A., Riley, R. L., and Lester, C. W.: Follow-up Study of Cardiopulmonary Function in Four Young Individuals After Pneumectomy, *J. THORACIC SURG.* 16: 30-49, 1947.
6. Johnson, Julian, Kirby, C. K., Lazatin, C. S., and Cocke, J. A.: The Use of a Prosthesis to Prevent Overdistention of the Remaining Lung Following Pneumectomy: a Preliminary Report, *Surgery* 22: 179-190, 1947.
7. Wilson, D. A.: Extrapleural Pneumonolysis With Lucite Plombage; a Preliminary Report, *J. THORACIC SURG.* 17: 111-122, 1948.
8. Small, J. T., Clagett, O. T., and Grindlay, J. H.: Additional Use of Gelatin Foam in Thoracic Surgery: Preliminary Report, *Proc. Staff Meet., Mayo Clin.* 22: 585-589, 1947.
9. Brown, M. H., Grindlay, J. H., and Craig, W. McK.: The Use of Polythene Film as a Dural Substitute; an Experimental and Clinical Study, *Surg., Gynec. & Obst.* 86: 663-668, 1948.
10. Seybold, W. D., Grindlay, J. H., Pfuetze, K. H., and Clagett, O. T.: The Use of Polythene Film in Posterior Thoracoplasty Wounds, *J. THORACIC SURG.* 18: 129, 1949.
11. Grindlay, J. H., and Mann, F. C.: Surgical Uses of Polythene: an Experimental Study, *Arch. Surg.* 56: 794-812, 1948.

THE ROENTGENOLOGIC MANIFESTATIONS OF PARASTERNAL OMENTAL HERNIA

JOHN S. STEWART, M.D.
COLUMBUS, GA.

THE preoperative diagnosis of parasternal omental hernia is usually based on the radiographic demonstration of a density in either cardiophrenic angle and cephalad displacement of a portion of the transverse colon. We wish to emphasize the use of a pneumoperitoneum as an accessory diagnostic aid.

The differential diagnosis of masses at the cardiophrenic angle involves consideration of the following: lymphomas, teratomas, lipomas, neurofibromas, aberrant thyroids, colonic cysts, and peripheral lung neoplasms. Our recent experience with the following three cases demonstrates the application of various roentgen procedures and may serve to evaluate their merits.

Parasternal omental hernias occur relatively infrequently. In 1937, von Gryerz,⁵ discovered in the literature six cases diagnosed during life by roentgenogram. Colmers,¹ in 1941, reviewed the literature and found 120 cases of various types of parasternal hernias. These included the six cases mentioned here. He found one case of parasternal omental hernia in which the diagnosis was suggested by chest roentgenogram and later proved by exploration. This case was reported by Meade and Ravdin.¹⁰

CASE REPORTS

CASE 1.—J. B. L., a white woman, aged 50 years, was admitted to Emory University Hospital in September, 1946. Chief complaints were fullness in the right side of the chest and a productive cough. The past history revealed a clinical diagnosis of pulmonary tuberculosis in 1918. Following hospitalization for two years, the patient was discharged with arrested disease. On a chest roentgenogram made in 1942, a small mass in the right lower lung field was discovered. This density subsequently increased in size. The physical findings referable to the chest consisted of dullness to percussion over the right lung base and absent breath sounds over the same area. Roentgenologic studies on admission showed the presence of a mass measuring about 8 cm. in diameter at the right cardiophrenic angle anteriorly (Fig. 1). A bronchogram disclosed separation of the right middle and lower lobe bronchi with partial obstruction of the secondary branches of the right middle lobe bronchus. A barium enema performed elsewhere revealed cephalad displacement of the transverse colon anteriorly with this portion of the colon having the appearance of an inverted "U" (Fig. 2). On bronchoscopy, separation of the right middle and lower lobe bronchi was observed.

An exploratory thoracotomy revealed a hernia of omentum through the right foramen of Morgagni with the sac presenting itself between the middle and lower lobes of the right lung. The hernia consisted of a peritoneal sac with partial incarceration of omentum.

CASE 2.—A. A., a white woman, aged 68 years, was admitted to Emory University Hospital in February, 1947, complaining of nervousness. One week previously, a routine roentgenogram of the chest showed the presence of a mass in the right cardiophrenic angle.

From the Department of Roentgenology, Emory University School of Medicine.
Received for publication Dec. 10, 1948.



Fig. 1.

Fig. 1 (Case 1).—Mass in right cardiophrenic angle.



Fig. 2.

Fig. 2 (Case 1).—Cephalad displacement of transverse colon as a result of omental hernia.

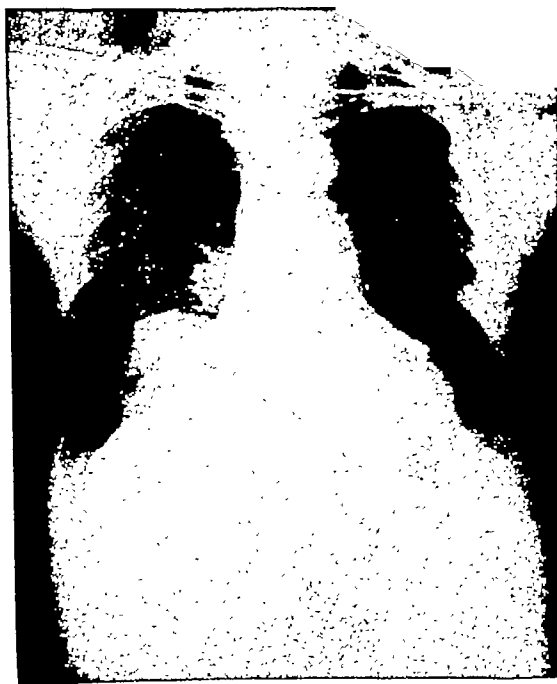


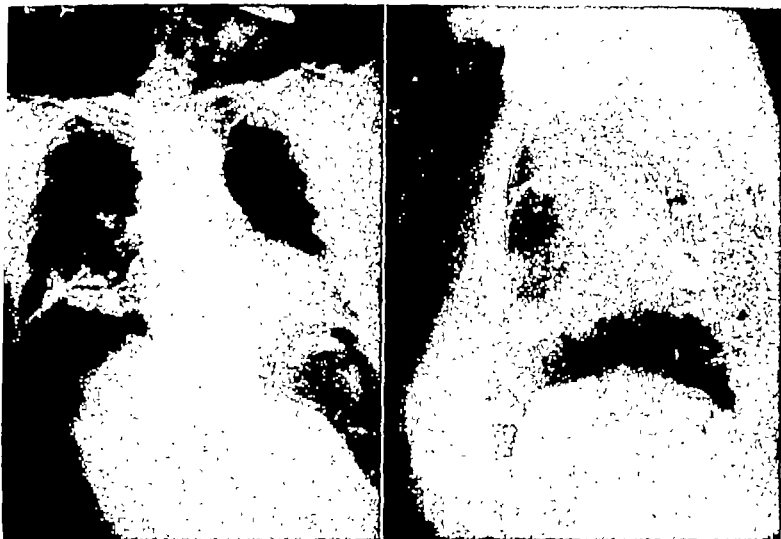
Fig. 3.

Fig. 3 (Case 2).—Mass in right cardiophrenic angle.



Fig. 4.

Fig. 4 (Case 2).—Cephalad and anterior displacement of transverse colon due to para-sternal omental hernia.



A.

B.

Fig. 5, A and B (Case 2).—Posteroanterior and lateral roentgenograms of the chest following introduction of pneumoperitoneum. Note air in hernial sac.



Fig. 6.

Fig. 6 (Case 3).—Lateral roentgenogram of chest and abdomen. Note mass in anterior costophrenic sulcus.



Fig. 7.

Fig. 7 (Case 3).—Parasternal omental hernia. Colon and stomach in normal position.

Physical findings referable to the chest were occasional râles over the middle lobe of the right lung. Sputum studies and bronchoscopy revealed no additional information. Roentgenologic examination of the chest showed a large oval-shaped mass at the right cardiophrenic angle anteriorly (Fig. 3). A lateral film of the abdomen demonstrated a gas-filled loop of colon which was displaced cephalad and anteriorly beneath the dome of the right diaphragm. This finding was confirmed by a barium enema (Fig. 4). Following the introduction of a pneumoperitoneum, air was demonstrated within the right cardiophrenic angle soft tissue mass (Fig. 5, *A* and *B*). A tentative diagnosis of parasternal omental hernia was made from these findings, and the patient was discharged without surgery because of age and absence of symptoms.

CASE 3.—A. O., a white woman, aged 50 years, was admitted to Emory University Hospital in December, 1947, complaining of pain in the right side of the chest and a productive cough of three months' duration. Two months prior to admission, a roentgenogram of the chest showed an area of increased density at the right lung base compatible with the diagnosis of a virus pneumonitis. Additional roentgen study two weeks later showed no change in the appearance of the lung fields. On physical examination, definite râles were heard at the right lung base. Bronchoscopy, sputum, and blood studies revealed no additional information.

Roentgenologic examination of the chest showed a soft tissue mass at the right cardiophrenic angle anteriorly (Fig. 6). Compression of the secondary branches of the right lower lobe bronchus was observed from bronchography. On barium enema, the colon assumed a normal position (Fig. 7). X-ray pictures taken after pneumoperitoneum showed no air within the intrathoracic mass.

An exploratory thoracotomy revealed a hernia 5 by 8 cm. between the middle and lower lobes of the right lung, which consisted of omentum in a peritoneal sac. The neck of the sac was obliterated by dense adhesions. The mass presented through the foramen of Morgagni on the right.

The patient made an uneventful recovery and now has no symptoms referable to the chest.

DISCUSSION

The embryology, anatomy, etiology, and classification of parasternal omental hernias have been discussed in detail by Colmers¹ and others,^{2, 3} and no attempt will be made here to enlarge upon their work.

Robbins¹¹ and Harrington^{6, 7} have emphasized the importance of the change in position of the transverse colon in parasternal omental hernias. With barium enema, the transverse colon assumes an inverted "V" pattern with the apex pointing toward the foramen of Morgagni. In one of the cases the transverse colon appeared less angulated and assumed the contour of a "U."

It has been found that the abnormal position of the gas-filled transverse colon may be suspected on a lateral roentgenogram of the abdomen. This finding is shown to advantage in Fig. 8.

Sielman,¹⁴ in 1924, demonstrated the presence of a hernial sac containing colon above the diaphragm utilizing pneumoperitoneum. He was able to differentiate the wall of the hernial sac from the wall of the large intestine. A small pneumothorax complicated this diagnostic procedure. Hayer,⁸ in 1933, warned of the complications of diagnostic pneumoperitoneum. These consisted of peritoneal adhesions and gangrene of the stomach with perforation from kinking of gastric arteries. In our experience, no complications resulted from the use of this procedure.

Sante,¹² Carman,² and Faulkner³ have all suggested the use of pneumoperitoneum in the diagnosis of subdiaphragmatic hernias. We have been unable to find in the American literature available to us a reference to the specific use of pneumoperitoneum in the diagnosis of congenital parasternal hernias containing omentum.

In Case 1, the abnormal position of the transverse colon was present, suggesting the correct diagnosis. In Case 2, the diagnosis was established without any doubt by pneumoperitoneum, although the characteristic sign of the barium enema might have sufficed.



Fig. 8 (Case 1).—Lateral film of chest and abdomen showing the abnormal position of the gas-filled transverse colon.

Of considerable importance is Case 3. In this patient none of the roentgen criteria used in Cases 1 and 2 gave evidence of the correct diagnosis.

There may be cases in which, in spite of negative barium enema studies, a pneumoperitoneum will reveal positive information. It is believed that, in these cases, pneumoperitoneum will have its greatest usefulness.

SUMMARY AND CONCLUSIONS

1. Certain roentgenologic studies are invaluable aids in the diagnosis of parasternal omental hernias. It is essential that these benign lesions be differentiated from neoplasm.

2. The roentgenologic findings may be as follows: (a) a soft tissue density at either cardiophrenic angle; (b) the change in position of the gas-filled transverse colon on a conventional lateral film of the abdomen; (c) the cephalad dis-

placement of the barium-filled colon assuming the contour of an inverted "V" or "U"; (d) the presence of air within the soft tissue mass following pneumoperitoneum.

3. There are certain cases in which none of the characteristic roentgen findings are present, making a definite preoperative diagnosis impossible.

REFERENCES

1. Colmers, Rudolph A.: Parasternal Diaphragmatic Hernia With a Report of a Case on the Right Side, *Radiology* 37: 733-739, 1941.
2. Carman, R. D., and Fineman, S.: The Roentgenologic Diagnosis of Diaphragmatic Hernia With a Report of Seventeen Cases, *Radiology* 3: 26, 1924.
3. Faulkner, W. B., Jr.: Diaphragmatic Hernia and Eventration, *Am. J. Roentgenol.* 45: 72-73, 1941.
4. Gale, J. W., and Edwards, J. W.: Malignant Tumors of the Diaphragm, *J. THORACIC SURG.* 9: 185, 1939.
5. von Gryerz, Walo: On Hernia Diaphragmatica Retrosternalis, *Acta radiol.* 18: 428-437, 1937.
6. Harrington, Stuart W.: Subcostalsternal Diaphragmatic Hernias, *Surg. Gynec. & Obst.* 73: 601-614, 1941.
7. Harrington, Stuart W.: Roentgenological Considerations in the Diagnosis and Treatment of Diaphragmatic Hernia, *Am. J. Roentgenol.* 40: 185-198, 1943.
8. Hayer, E.: Seltene Formen von Zwerchfellhernien und ihre röntgenologische Erfassung, *Fortschr. a. d. Geb. d. Röntgenstrahlen* 48: 165-176, 1933.
9. Kirklin, B. R., and Hodgson, John R.: Roentgenological Characteristics of Diaphragmatic Hernia, *Am. J. Roentgenol.* 58: 77-100, 1947.
10. Meade, R. H., and Ravdin, I. S.: Nontraumatic Hernia Through the Right Parasternal Foramen of Morgagni, *Ann. Surg.* 102: 465-471, 1935.
11. Robbins, Lawrence L.: Roentgenological Diagnosis of Parasternal Omental Hernia, *Radiology* 41: 378-382, 1943.
12. Ruschioni, M.: Un caso di ernia diaframmatica parasternale, *Radiol. med.* 14: 1116-1123, 1927.
13. Sante, L. R.: Pneumoperitoneum as an Aid in the Diagnosis of Subdiaphragmatic Conditions, *J. A. M. A.* 80: 464-466, 1923.
14. Sielman, H.: Ein Fall von Hernia Diaphragmatic Parasternalis vera, *Fortschr. a. d. Geb. d. Röntgenstrahlen* 32: 426-430, 1924.

AORTOESOPHAGOGASTRIC FISTULA, AN UNUSUAL COMPLICATION OF ESOPHAGOGASTROSTOMY PERFORMED UNDER THE AORTIC ARCH FOLLOWING ESOPHAGEAL RESECTION FOR CARCINOMA

A REPORT OF TWO CASES

K. ALVIN MERENDINO, M.D.,* AND E. C. EMERSON, M.D.
MINNEAPOLIS, MINN.

ESOPIHAGEAL resection for carcinoma with the restoration of the continuity of the gastrointestinal canal now is a well-established procedure. The mortality of this procedure is related to (1) the older age of the patients afflicted with this disease, (2) the problem of anesthesia, (3) the location of the lesion with regard to its position above or below the aortic arch, (4) the technical difficulties associated with any anastomosis of the esophagus, and (5) the complications of infection within the thoracic cage.

It is apparent that certain of the complications which have resulted in a rather high mortality rate must be considered as unavoidable because of the age of the patients combined with a disadvantageous location of the lesion. At the present time the morbidity and mortality are increased markedly when the resection and anastomosis must be performed above the arch of the aorta. Undoubtedly, as more experience is gained with the higher lesions, a better knowledge of the most satisfactory method will be the result.

The complications which must be considered as being avoidable are related mainly to the problems of technical difficulties, anesthesia, and infection, either primary or secondary to trauma and/or leakage in the area of the anastomotic line. Although the antibiotics have altered the prognosis of infection in general, the problem of its avoidance in the chest still is of paramount importance.

The recent observation of two cases exhibiting an unusual complication following esophagogastrostomy has prompted this presentation. No previous report has been found describing a similar complication. In both instances, the patient died of an exsanguinating gastrointestinal hemorrhage from an aortoesophagogastric fistula.

CASE REPORTS

CASE 1 (No. 763980).—H. S., a 63-year-old man, was admitted to the University Hospital on April 9, 1946, and died on May 4, 1946.

History.—The patient gave a history of dysphagia, regurgitation, and epigastric distress for the previous three months. There had been a twenty-pound weight loss in the past month. Immediately prior to admission, he was able to take only liquids augmented by some soft food. There was a history of two previous cerebral accidents having occurred thirteen and seven years, respectively, prior to admission.

Physical Examination.—Examination revealed a somewhat asthenic, deaf, white man with a right hemianopsia and a right hemiplegia. The blood pressure was 182/100, respirations were 22, and pulse was 80.

Laboratory Findings.—The hemoglobin, white blood cells, urine, Kline and Kahn, blood urea nitrogen, plasma protein, venous pressure, and vital capacity were either negative or within normal limits.

From the Department of Surgery, University of Minnesota.

Received for publication Jan. 11, 1949.

*Now at the Department of Surgery, University of Washington School of Medicine, Seattle, Wash.

On March 21, 1946, a gastrointestinal study revealed a carcinoma of the lower mid-portion of the esophagus.

A roentgenogram of the chest revealed a nodular density in the left base, possibly representing a metastatic nodule. There was a small area of pneumonia in the right middle field.

The electrocardiogram was thought to be abnormal. No definite diagnosis was suggested on the basis of this examination.

The patient was prepared for surgery by the oral route for a period of thirteen days by means of a liquid high protein, high carbohydrate, high caloric diet.

Operation.—On April 22, 1946, the patient was taken to the operating room for an exploratory thoracotomy on the left side, through the bed of the fifth rib, which was removed.* The carcinoma was noted to begin approximately 2 or 2½ cm. below the inferior border of the arch of the aorta. This had perforated into the adjacent tissues. It was felt that there were probably metastases in the hilus of the left lung and some metastatic nodules along the superior border of the pancreas in the abdomen. The lesion was entered into in the course of dissection, and the operator was committed to excision of the lesion. An esophagogastrotomy was performed just at the inferior border of the arch of the aorta without undue tension. Interrupted silk sutures were used. The diaphragm was sutured about the stomach.

Pathologic Report.—

Gross: The specimen consisted of a segment of esophagus measuring 8 by 5 cm. The mucosal aspect presented an ulcerated nodular elevation. The edge of the lesion extended to within 3 mm. of the proximal line of resection.

Microscopic: The tumor was composed of poorly differentiated, squamous epithelial tumor cells growing in irregular masses and clumps; in the center of some, necrosis was present, and others showed only cornification. The proximal and distal lines of resection were free of tumor. The tumor had penetrated all layers of the esophagus and was growing on the surface of the specimen. No lymph nodes could be found.

The conclusion was a squamous-cell carcinoma of the esophagus.

Postoperative Care.—The patient's temperature varied from 99 to 103.2° F. with a corresponding increase in pulse rate without any remarkable increase in the respiratory rate. A roentgenogram on April 29, 1946, revealed a small residual pleural effusion in the left base. On May 2, 1946, the chest examination revealed minimal infiltration on the upper lobe on the right. On physical examination on this day, a pleural friction rub was heard over the chest anteriorly. At 6:55 A.M. May 4, 1946, the patient experienced a sudden onset of dyspnea, cyanosis, pain in the chest, and apprehension. Almost immediately he had a small "hemoptysis." A subsequent "hemoptysis" occurred at 8:15 A.M. Some pulmonary edema was present. The blood pressure was 100 systolic, and the examiner was unable to determine the diastolic pressure; the pulse was 120. The impression at that time was large pulmonary infarct, although there were no signs of thrombophlebitis or phlebothrombosis of the lower extremities. At 8:30 A.M. a bilateral cervical sympathetic block was performed with 1 per cent novocain. The patient died approximately fifteen minutes later.

Autopsy Examination.—At the point of anastomoses there was a fistula between the aorta and esophagogastric anastomosis apparently created by a suture which passed through the media of the aorta and the esophagogastric anastomosis. This had resulted in a large hemorrhage from the aorta through this fistula into the stomach and bowel. This blood extended up behind the esophagus into the neck, behind the great vessels and along the paravertebral spaces. The remainder of the gastrointestinal tract contained approximately 3,000 c.c. of fresh blood. This figure of 3,000 represented the measured amount of blood. In addition, there was a large volume of blood which poured out of the bowel when it was opened. Examination of the pancreas revealed it to be hard and indurated along the superior border where it was matted to several celiac nodes.

The examination of the brain revealed: (1) cerebellar arteriosclerosis and (2) old encephalomalacia.

The conclusions were: (1) metastatic squamous-cell carcinoma of the celiac nodes with extension into the pancreas; (2) an aortoesophagogastric fistula with an extensive hemorrhage from the aorta through the anastomotic line into the gastrointestinal tract and into the mediastinum.

*Operation by a colleague, assisted by K. A. M.

CASE 2 (L-1063).—R. L., a 50-year-old white man, was first admitted to the St. Johns Hospital, St. Paul, Minn., and was transferred to the University Hospital on Nov. 24, 1947, and discharged on Jan. 5, 1948.

History.—The patient gave a history of anorexia, some pain in the epigastrium, and minimal dysphagia of approximately six weeks duration. The patient had lost twenty-five pounds in this period.

Physical Examination.—The physical examination disclosed a somewhat emaciated adult man. The remainder of the examination was essentially negative.

Laboratory Findings.—The laboratory findings revealed a normal blood picture and urine. On an x-ray examination of the chest, the heart and lungs were found to be normal. A gastrointestinal study was performed, at which time a transient obstruction to the flow of barium in the esophagus at the level of the ninth and tenth dorsal vertebra was observed. There appeared to be a slight constriction of the esophagus in this area and a small mass on the posterior wall projecting into the lumen of the esophagus. The esophagoscopist reported a small granulating area at the point of the junction between the middle and lower third of the esophagus. A biopsy was taken.

The microscopic examination of this area showed infiltrating aberrantly growing masses of squamous epithelium.

The diagnosis was a squamous-cell carcinoma of the esophagus.

Operation.—The patient was prepared for surgery, and on Dec. 9, 1947, a left posterior transthoracic approach excising the sixth rib was performed (K. A. M. assisted by E. C. E.). No palpable nodes were present. The lesion in the esophagus was found at the junction of the middle and lower third of the esophagus and was the size and shape of a small lemon. The esophagus proximally was sacrificed immediately distal to and underneath the arch of the aorta. The distal line of resection was through the junction of the proximal one-fourth and distal three-fourths of the stomach. The left gastric artery was amputated at the site of its exit from the celiac axis. The distal stomach then was closed by running sutures supplemented by an additional row of 0000 Deknatel sutures. The spleen was sacrificed during the operative procedure. An esophagogastrostomy was performed in the usual fashion. Two catheters were placed in the chest, one posteriorly along the posterolateral gutter, and the other was placed at the site of the anastomosis. Care was taken to wrap the anastomosis with a substantial portion of the gastrosplenic ligament. This was not sutured in place.

Postoperative Course.—The early postoperative course was smooth; the highest elevation was 100.2° F. on the fourth postoperative day. The temperature then fell to normal the following day. During the early postoperative days difficulty was encountered in the expansion of the left lung. On the ninth postoperative day the temperature, having been normal for five days, rose to 102° F. By means of aspiration and the insertion of additional catheters in the chest under water-seal drainage, the lung finally re-expanded. On the seventeenth postoperative day a foul drainage was noted from the intercostal catheters. The presence of a fistula was suspected. Methylene blue given by mouth confirmed the diagnosis. The patient who had been eating six small feedings daily was taken off all feedings by mouth with the exception of one ounce every hour of distilled water containing 1,000 units of penicillin per cubic centimeter. Within three days the fistulous tract could be no longer demonstrated. The empyema cavity then was irrigated with Dakin's solution every four hours. The remainder of the convalescence was uneventful, and on Dec. 30, 1947, after the instillation of 10 c.c. Lipiodol, an open thoracotomy was established through the bed of the ninth interspace posteriorly. The patient was discharged six days after the last operative procedure.

Follow-up.—Within a short period of time the empyema cavity had healed and the thoracotomy tube was removed. The patient was able to take semisolid food without difficulty in swallowing. An x-ray examination on Jan. 14, 1948, revealed some slight area of narrowing at the point of anastomosis. There was evidence of a blind tract extending from the line of anastomosis laterally for approximately 1 cm. and approximately 3 or 4 mm. in diameter. Dilatation was carried on with the Vincent type dilator. A No. 21 French dilator was passed without difficulty. Subsequently, a No. 31 French followed by a No. 37 French dilator was passed without undue difficulty.

On Feb. 27, 1948, following a rather large dinner, the patient complained to members of his family of a rather sudden severe substernal pain and dyspnea and shortly became apprehensive. This was followed by a moderate-sized hematemesis. He was admitted immediately to the hospital.

Second Hospital Admission.—The patient was admitted at 8:45 P.M. on Feb. 27, 1948, and died at 1:00 P.M. on Feb. 28, 1948.

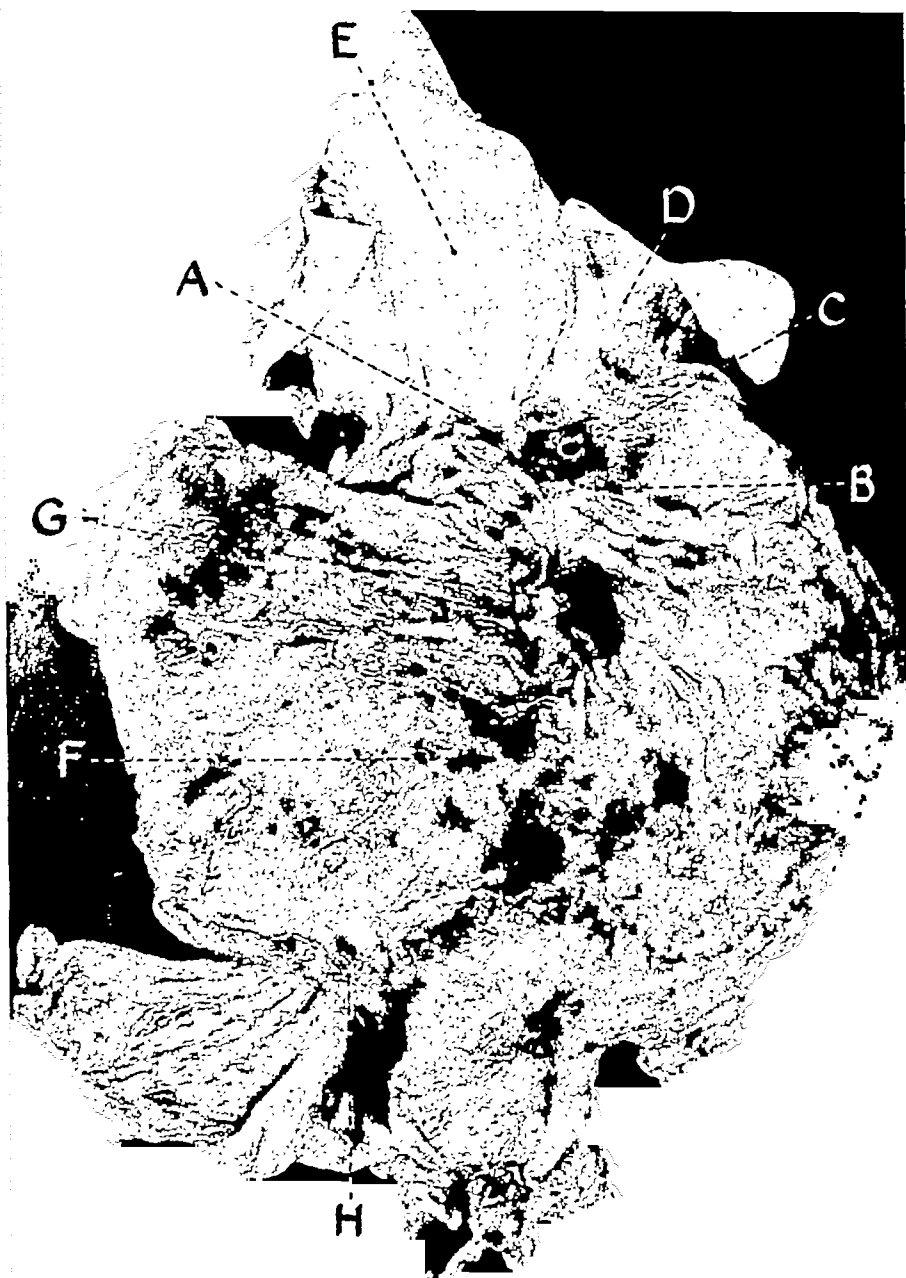


Fig. 1.—Autopsy specimen. The esophagus and stomach have been opened anteriorly in the midline. A, Anastomosis of the esophagus to the stomach; B, ulcer (probably represents a separation of the anastomosis) at the esophageal gastric anastomotic line; C, aorto-esophagogastric fistula; D, arch of the aorta; E, esophagus; F, residual stomach; G, line of inversion of the distal residual gastric pouch; H, pyloric sphincter.

The patient was entirely conscious upon admission. The blood pressure was 88/64, pulse 120, respirations were 24. Shock blocks were inserted under the foot of the bed, and morphine sulfate was administered. Periodically he continued to vomit varying amounts of bright red blood. (The largest measured amount was 1,300 c.c.) Although the patient appeared to be desperately ill, he continued to be conscious and responded well to questioning up until his death the following day at 1:00 p.m. The total amount transfused before death was 5,000 c.c. of blood, calcium having been given during the night.

Because of the previous experience of observing an aortoesophagogastric fistula following an esophagogastrostomy in this location, this diagnosis was entertained in this

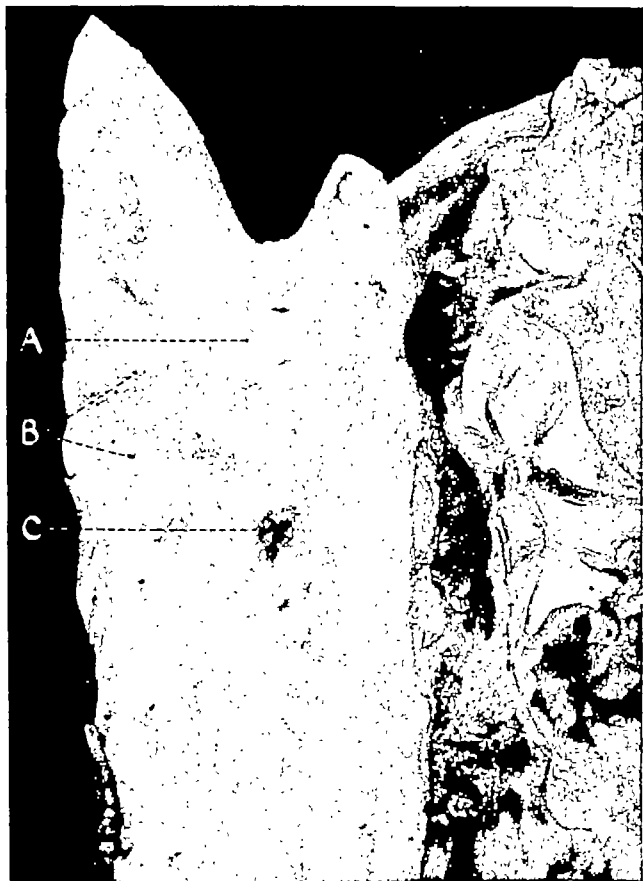


Fig. 2.—Autopsy specimen. The aorta has been opened posteriorly in the midline. A, Arch of the aorta; B, orifices of the intercostal vessels; C, aortoesophagogastric fistula.

patient. At no time during the patient's second admission to the hospital was he in a condition sufficiently satisfactory for an exploration of the site of the anastomosis to be contemplated.

Autopsy Examination.—At autopsy* an aorto-esophagogastric fistula was found which measured 6 mm. in diameter on the aortic side. Within the lumen of the esophagus and stomach corresponding in position with the aortic perforation, an ulcer (2 cm. in diameter) at the esophagogastric anastomosis on the left lateral side was found (see Figs. 1 and 2). There was no evidence of recurrent tumor. An incidental finding was the presence of a small area of irregular glands in the prostate which were histologically malignant. Microscopically, the ulcer contained acute and chronic inflammatory cells. The margin of the opening in the aorta was necrotic and also was infiltrated with inflammatory cells.

DISCUSSION

The proximity of the aorta to the line of anastomosis undoubtedly carries with it an additional hazard. If trauma to the aorta is avoided and primary healing of anastomosis occurs, this complication probably will not occur. In each of the cases presented, one of these factors played a role in the development of the aorto-esophagogastric fistula.

In the first case, the aorta was traumatized by a misplaced suture passing through the media of the aorta and the esophagogastric anastomosis. In the second case, in spite of a carefully performed completely satisfactory anastomosis, leakage occurred at the suture line (seventeenth postoperative day) with the development of an esophagogastric pleural fistula. This area almost immediately was sealed by the juxtapositioned aorta. It is conceivable that such a fistula might communicate with the main pulmonary artery, the inferior pulmonary vein, or the bronchial vessels. The major pulmonary vein is well protected by the interposed bronchus. The vessel involved, of course, would depend upon the level of the anastomosis. However, the danger of a venous erosion is less likely. It is well known that in the presence of infection because of the constant pulsation of an arterial vessel, perforation is more likely to occur in an artery than in a vein similarly exposed.

It is apparent that in order to protect the patient against the possibility of such a complication, it is important to avoid direct contact of the anastomotic line with any major vessel in the area. This can be accomplished readily by the interposition of a satisfactory tissue between these structures. In the second case, with the observation of the first catastrophe still fresh in mind, a large portion of the gastrosplenic ligament was spared in the abdominal dissection for the express purpose of wrapping it about the anastomosis. This was done. At the time of the autopsy the aorta was adhered to the esophagogastric anastomosis and, in addition, the wall of the aorta formed the base of the ulcer found at the anastomotic level. This finding suggests that either a dissolution of the gastrosplenic ligament took place or the position of this tissue was altered when the patient assumed the upright position on the first postoperative day. In retrospect, the choice of tissue was a poor one. The gastrosplenic ligament receives the major portion of its blood supply from the vasa brevia derived from the distal portion of the splenic artery. These arterial vessels usually are divided

*By Dr. John F. Noble.

when a concomitant splenectomy is performed and/or sacrificed when the stomach is mobilized for its craniad intrathoracic displacement. In order to obtain a tissue with an intact blood supply, a portion of the gastrocolic ligament which is well nourished by the right gastroepiploic artery should be utilized. Furthermore, in order to be assured that this tissue will remain in position when so placed, it should be sutured carefully about the esophagus and the retro-esophageal tissue. In this fashion any direct contact between major vessels in this area and the esophagus is obviated.

There are certain common features in these cases which should allow serious consideration of a diagnosis of aortoesophagogastric fistula. In each instance an esophagogastrostomy had been performed immediately distal to and under the arch of the aorta. This fact, combined with the subjective findings of sudden pain in the chest accompanied by dyspnea and apprehension, plus the objective findings of a varying degree of shock accompanied by hematemesis, should allow the presumptive diagnosis to be made. In the second case, there was no vomiting of blood in the usual sense but rather a regurgitation of the blood without writhing or any obvious effort.

Granted that the patient's condition can be improved sufficiently by massive transfusions to allow the consideration of a surgical approach, the arterial defect could be explored and undoubtedly, in certain instances, closed without great difficulty. Certainly there is everything to gain and nothing to lose by such a maneuver. In Case 2, although the tissue about the perforation was necrotic for approximately 1 mm. with acute and chronic inflammatory cells present, the consistency of the aorta 1 or 2 mm. peripheralward about the circumference of the fistula was of sufficiently good quality to allow a satisfactory closure of the defect.

Only by such an aggressive approach can the life of a patient who otherwise is doomed be salvaged.

SUMMARY

The unusual complication of an aortoesophagogastric fistula resulted in the death of two patients herein reported. Both patients had been subjected to esophageal resections for carcinoma with the restoration of the continuity of the gastrointestinal tract by an esophagogastrostomy distal to and underneath the arch of the aorta. Certain aspects concerning the avoidance of this complication have been discussed. The clinical features observed in these two cases have been presented. The consideration of this complication as a diagnostic possibility has been stressed. The feasibility of a surgical closure of the defect has been predicted.

ANEURYSM OF THE FIRST PART OF THE LEFT SUBCLAVIAN ARTERY

REVIEW OF THE LITERATURE AND A CASE HISTORY

LESLIE J. TEMPLE, F.R.C.S.

LIVERPOOL, ENGLAND

EVER since the second century, when Antyllus (works of Oribasius) first described what is now the modern surgical treatment of aneurysms, ligation of the first part of the left subclavian artery must have been in the nature of a challenge to surgeons. No one can fail to be thrilled by Halsted's words (1920):

In ligating the first portion of the left subclavian in the chest the operator may not, as formerly, be more greatly impressed by the magnitude and cleverness of his performance than by the miraculous effect of the ligation of the artery upon the great pulsating tumour which with each beat of the heart jarred the whole frame of the sufferer. The moment of tying the ligature is indeed a dramatic one. The monstrous booming tumour is stilled by a tiny thread, the tempest silenced by the magic wand.

It is true that many men revered in surgery have condemned the operation as impossible. On reading the accounts of earlier operations, particularly those of the pre-Listerian era, this attitude can be understood. Of the 21 cases collected by Halsted up to 1920, there were 8 patients who died and only 8 were known to be cured. In only 2 patients did the aneurysm originate as far medially as the junction of the first and second parts of the artery. In all except 1 the ligation was undertaken from above and anteriorly by dividing the sternomastoid after resecting a portion of the clavicle and sometimes resecting the manubrium and even one or two inches of the first rib. Even in the patients who recovered, the morbidity was high, due to damage to the thoracic duct or the pleura or sepsis resulting on tamponade for uncontrollable bleeding. Halsted's critical comments on each of the cases described are still invaluable, but although when he wrote his monograph Morrision Davies, Sauerbruch, and others had shown that the pleural frontier could be violated with impunity, the idea of a transpleural ligation does not seem to have occurred to him. In only one of the cases he describes was a different approach used. Sherrill (1911), operating for what he believed to be a subclavian aneurysm, resected three inches each of the second, third, and fourth ribs posteriorly and approached the mediastinum extrapleurally. He found it easy to ligate the subclavian, but unfortunately the aneurysm originated in the aortic arch.

Lee, Mitchell, and Peacock (1934) described a successful operation by the superior approach for a traumatic aneurysm of the first part of the left subclavian artery. From the literature they were able to collect 128 cases of traumatic aneurysm of a subclavian artery since 1767, including Halsted's and

From the Liverpool Thoracic Surgical Centre, Broadgreen Hospital.
Received for publication Feb. 11, 1949.

Souchon's (1895) collected cases. The only additional cases, however, in which quite indisputably the first part of the left subclavian was ligated, were those of Von Haberer and their own.

Von Haberer (1916) operated on 72 patients with aneurysm due to gunshot wounds, 7 of which were in the left subclavian artery. Four of these involved the first part of the artery and were exposed by division of the clavicle. In 2 patients, after excision, an anastomosis of the arterial ends was performed. There was one death in this series. The remaining 3 patients were dealt with by supraclavicular incisions, with an anastomosis in one.

Kirchner's (1927) case was due to a wound received in 1917, and the patient was operated upon in 1927 by a supraclavicular route.

A search of the literature since then reveals comparatively few cases, except those associated with cervical ribs. These, I feel, come into a separate category, partly because the aneurysm always forms distal to the point of compression (MacFec, 1940) and is thus readily accessible to the surgeon, and also because the condition is almost always relieved by removal of the offending rib. Lindsborg and Howes (1937), in addition to reporting a case, reviewed the literature of aneurysms associated with cervical rib and reached similar conclusions.

An important step forward was made by Henry (1923) who, from the dissecting room, described a posterior mediastinal approach to the first part of the left subclavian. The soft tissues are turned up as a medially hinged flap, and the back end of the second rib and the transverse process are resected. The pleura is stripped from the mediastinum and the origin of the artery exposed. He quoted Sherrill's case as the only patient actually operated upon by posterior mediastinotomy.

Wright Smith (1927) reported a patient with syphilitic aneurysm of the second and third parts of the left subclavian operated upon by Hailes. Hailes resected the posterior ends of the second and third ribs to reach and ligate the first part of the artery. Thus, although Wright Smith called it a Henry operation, it was much more like Sherrill's original approach.

Nelson (1932) quoted a case by Alexander, who approached a right subclavian aneurysm by a similar route, but on this side it was less successful, and after a subsequent operation from the front to ligature the innominate artery, the patient died. Incidentally, during the posterior mediastinotomy, the pleura was unintentionally widely opened.

Henry's approach, however, found approval. Matas (1926), reviewing twenty-five years experience, mentioned six ligations of the first part of the left subclavian for aneurysm, only two being intrathoracic. The approach was always the classical one from above and in front, but he concluded by recommending Henry's operation. Similarly Elliott (1932), although he described no relevant case in his discussion on subclavian aneurysm, commended Henry's approach.

Muller (1935) described a traumatic aneurysm of the second and third part of the left subclavian. He ligated the first part of the artery by the anterior extrapleural route and subsequently successfully excised the tumor. He reviewed the literature and discussed the subject. Worth mentioning is his comment from

Elliott's earlier paper (1912), that for aneurysm of the third part of the subclavian the first part of the artery should be ligated as a preliminary. In view of the case now to be reported, two points in his paper are particularly significant. After careful stripping of the pleura in the anterior approach, it became torn, but no ill effects resulted. In his discussion he stated that if the aneurysm was in the first part of the left subclavian no treatment was possible except distal ligation.

The literature of World War II does not seem to include many relevant cases. Adams (1946) reported six subclavian aneurysms but did not give details. In each case the approach was through or above the clavicle.

Schumacker and Carter (1946), reviewing arteriovenous fistulas and arteriovenous aneurysms in military personnel, described 364 cases. These included 10 subclavian fistulas and 5 aneurysms with 1 operative death in the latter group. Side and site of the lesions were not stated nor was the operative approach described.

Elkin (1946) discussed 450 cases of aneurysms and arteriovenous fistulas in military personnel. In 45 patients part of the clavicle was resected but these included carotid, innominate, subclavian, and axillary aneurysms, with proportions not stated. Significantly, the only death was in a patient in whom the first part of the left subclavian was affected, and was due to failure to get a suture on the proximal end of the artery through the exposure used.

An anterior combined thoracic and cervical approach to the left subclavian was described by Wilson and Carr (1948). In a case of arteriovenous aneurysm they divided the sternomastoid, removed the medial three-quarters of the clavicle, divided all the ribbon muscles, and split the sternum to the level of the third rib and cut across laterally to aid retraction. They then proceeded with an extra-pleural exposure and successful treatment of the fistula.

Freeman (1946), describing 23 cases of aneurysm and fistulas due to war wounds, included an arteriovenous fistula of the left subclavian and the left innominate vein. The operative approach included division of the sternomastoid and strap muscles, resection of one-half the clavicle, and splitting of the sternum to the second cartilage. A successful repair was effected, but some wound infection necessitated reopening of the wound. The final outcome was satisfactory.

Rehn's cases (1942) appeared to concern the third part of the artery only. Other authors with extensive experience of war wounds of the chest supplied no relevant cases. These include Miscall and Harrison (1947) who reported 300 chest wounds, Martin (1947) who did follow-up on 106 cases for three months, Snyder and Tropea (1947) who reported 678 cases, and Tudor Edwards (1943) who, in his survey of war wounds and injuries to the chest, did not mention this particular injury. It is concluded that wounds involving the first part of the left subclavian resulted in death and not aneurysm.

For the sake of completeness it should be added that two articles which have been published in the Russian journals and two others by Bsteh (1947) and Perrotti (1947) could not be obtained, and it is not known whether or not the subclavian aneurysms which they described are relevant.

A search of the records of the Liverpool and North West Regional Chest Surgical Unit since 1940 revealed only one case of aneurysm of the first part of the left subclavian artery. This patient, a soldier 28 years of age, who sustained a gunshot wound of the left axilla at Anzio in February, 1944, was operated upon by Mr. H. Morriston Davies in June, 1944. The foreign body responsible for the aneurysm was accurately localized by roentgenogram and the chest opened by an incision into the third interspace from in front with division of the cartilages. The pleura was opened and the adherent lung freed. A nonpulsating swelling was found at the junction of the left subclavian and aorta corresponding to the site of the metallic foreign body. A needle was inserted into the swelling but only fibrinous fluid was obtained. A second needle was then inserted and blood under pressure forced up the piston of the syringe. At the same time blood began to spurt from the first needle hole. Both holes were oversewn and the chest closed. The patient suffered no ill effects but a roentgenogram one month later showed an increase in the size of the aneurysm. He was transferred to a Ministry of Pensions hospital and no further records are available. This case has been quoted in full, as it is the only one I have found recorded in which a direct intrapleural approach to the left subclavian artery has been made.

The report of my own case follows.

The patient was an engine driver, aged 40 years, who was admitted to the Liverpool Chest Surgical Centre on Aug. 3, 1948.

The history given was that he became ill with pneumonia of the left lung on May 8, 1948, and spent six weeks in bed. At the end of that time he was quite well, without cough or dyspnea, or pain in the chest. His own doctor examined him before sending him back to work and found abnormal physical signs, as a result of which roentgenograms were made and eventually the patient was sent to the Chest Unit. He had no complaints whatsoever at the time and was anxious to get back to work.

On examination he was a spare but healthy looking man, rather young for his age. The most striking feature was a fully developed left Horner's syndrome (Fig. 1). After questioning about this, he stated that in February, 1946, he was knocked down by a railway engine and fell striking his chest on a projection on the ground. He spent ten days in the hospital after this accident, but roentgenograms were not made; on discharge he noted a difference in the size of his eyes for the first time. The condition had never worried him greatly and had not altered in appearance since. There was no sign of finger clubbing in either hand, but the left hand was definitely warmer than the right. Radial pulses were equal.

On examination the upper left part of the chest, back and front, was stony dull to percussion, and breath sounds were absent. No other abnormality whatsoever was noticed. A roentgenogram of the chest showed a large rounded tumor extending from the apex of the thoracic cavity down to the seventh rib posteriorly and reaching medially to the mediastinum. This opacity was clearly defined and rounded. The rest of the lung fields were clear. (See roentgenograms illustrated in Figs. 2 and 3.) The tumor did not pulsate on screening.

Blood count was within normal limits. Wassermann reaction and Meinicke were negative, and a Casoni test proved to be negative. Sputum was mucoid and contained buccal flora only.

I had not much doubt that this was a case of neurinoma, of some type, either originating from one of the intercostal nerves or, in view of the sympathetic paralysis of the eye and left upper limb, originating from the sympathetic chain as a ganglioneuroma. If the former, there was of course the possibility of an extension into the spinal canal; therefore, I asked

Mr. R. H. Hannah of the Liverpool Neuro-Surgical Unit to see the patient, and he agreed that there was no direct involvement of the central nervous system and he was prepared to be present at the operation and undertake the removal of any part of the tumor that entered the spinal canal.



Fig. 1.—Photograph of the patient showing the ptosis enophthalmos and small pupil, left side.



Fig. 2.

Fig. 2.—Posteroanterior roentgenogram of the chest.

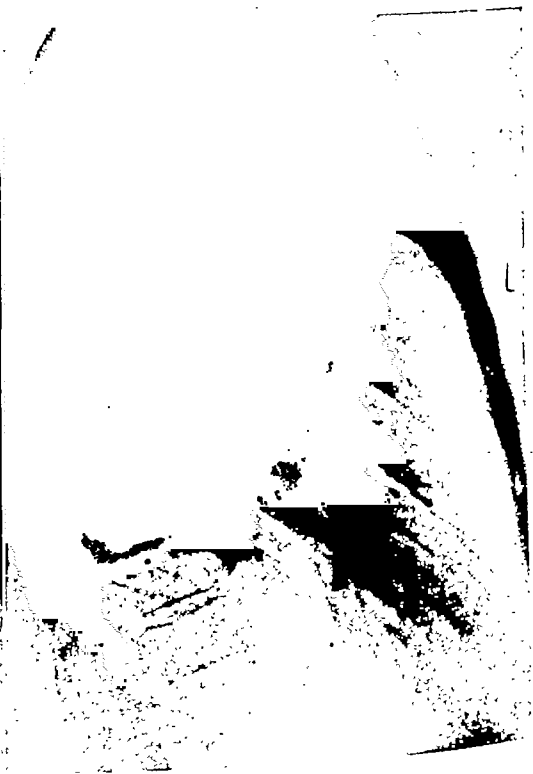


Fig. 3.

Fig. 3.—Lateral roentgenogram of the chest.

Operation was undertaken on Aug. 25, 1948. Anesthesia consisted of Tubarine, Pentothal, and nitrous oxide*; a blood transfusion was started running slowly in the saphenous vein before the operation was begun. The patient being in the lateral position, the fifth rib was resected and the fourth and third ribs divided posteriorly. The pleura was opened, but the pleural space was almost completely obliterated by adhesions, and the lung was separated only with considerable difficulty. A large tumor in the apex of the chest was closely adherent to the lung itself and could be separated only by sharp dissection, damaging the lung somewhat in the process. The tumor was further defined once the lung was freed and on the posterior surface stripped extrapleurally. It was found to be attached to the first thoracic nerve at its origin and was hard, not pulsatile, but somewhat cystic to feel. On medial dissection the subclavian artery appeared to run into the substance of the tumor within one inch of its origin from the aortic arch and only left it at the inner edge of the first rib. It was still not certain whether this was adherence or whether the tumor was an aneurysm (Fig. 4).

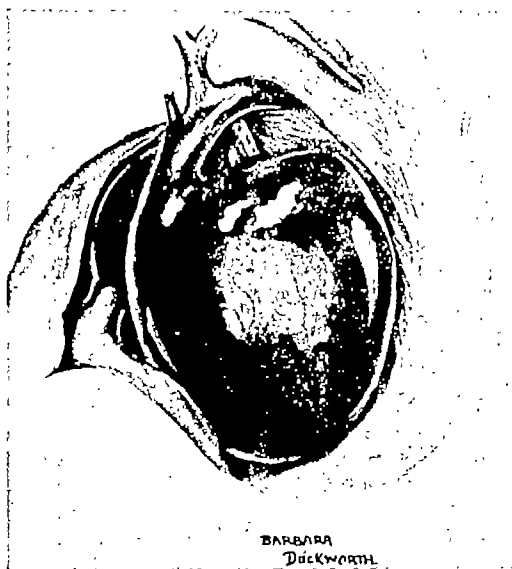


Fig. 4.—The tumor as seen at operation.

A purse-string suture was placed around one point of the aneurysm and a needle inserted.

One hundred cubic centimeters of brownish altered blood were aspirated, after which the needle was withdrawn and the purse string tied. This produced some diminution in the tension of the cyst for the time being, but it soon became as tense as before, and during the later dissection, the purse string opened and this time bright red blood spouted from the needle puncture. Before proceeding further a loose thread was put around the origin of the subclavian and one at the inner border of the first rib. At this stage the subclavian was digitally compressed against the first rib, and the appearance of his left hand was noted. It was somewhat cyanosed, but the skin circulation seemed to persist. No radial pulse was palpable. It now seemed clear that the tumor was an aneurysm, and an attempt was made to separate it from the subclavian artery and narrow its pedicle. A

*Administered by Miss Noel Fenton.

large number of small vessels were dealt with and hemorrhage controlled, until on further separation of the aneurysm from the wall of the artery a very copious hemorrhage ensued. Traction on the two ligatures that had been passed around the subclavian failed to control it, so rubber-covered clamps were applied to the artery at the same points. The bleeding still occurred as soon as the compressing finger was removed. Later I found that one of the rubber-covered clamps was defective and it seems this was the cause of the failure to control hemorrhage by this method. Clamps were then applied directly to the bleeding part of the artery, the aneurysm was separated from it and dissected away leaving a considerable gap in the side of the subclavian artery. The mass stripped quite easily off the first thoracic nerve to which it was attached only by fibrous tissue. The subclavian artery was ligated and then transfixed one-half inch from its origin on the aortic arch and again immediately below the origin of the internal mammary artery. The intervening portion of the damaged artery was removed. The chest was closed with an underwater-seal drain. Examination of the sac showed it to be almost filled with laminated blood clot, and it was reported on by the pathologist* as follows: A cyst about 4 inches in diameter was received containing blood. The thin part of the wall consisted of compressed acellular connective tissue. A small thick portion showed organized clot into which new vessels were growing. The wall of this part was of chronically inflamed fibrous tissue, in which were identified blood vessels, nerves, and some islands of elastic tissue. The appearances were consistent with the diagnosis of aneurysm.

At the conclusion of the operation no left radial pulse was palpable, and the blood pressure could not be measured in the left arm. The hand was somewhat cyanosed, but the skin circulation was reasonably brisk. Nursing care included keeping the body as warm as possible, with the arm lying horizontally and at rest outside the bedclothes. At no time did he give rise to any anxiety whatsoever, and we could not stop him using the arm for lighting cigarettes, etc., after the first postoperative day.

Three weeks after operation he continued to have no left radial pulse, but there were no sensory changes and no loss of power in the arm. The cyanosis had disappeared and the hand remained warmer than the other one. The patient was then given permission to return to his job, which he was very anxious to do. Most surprising of all, there had been a definite diminution in the ptosis of the left eye, although the pupil remained small.

DISCUSSION

Discussion centers on two aspects of the case, diagnosis and operative treatment.

Preoperative diagnosis was missed largely because insufficient attention was paid to the history. The history is given as it was received, and there was a tendency to dissociate the accident from the appearance of the Horner's syndrome, assuming that the stay in the hospital merely drew the patient's attention to a pre-existent state. Had the history been presented simply as that of an accident followed immediately by the appearance of Horner's syndrome and thereafter the discovery of an intrathoracic tumor, it is more likely that the diagnosis of aneurysm would have been reached. The failure to detect pulsation on screening is not surprising. The aneurysm in Mr. H. Morriston Davies' case did not pulsate, and Kerly (1939) stated that only 25 per cent of intrathoracic aneurysms show expansile pulsation on screening. The fact that there was no palpable tumor or pulsation in the neck is more significant. None of the cases in the literature reviewed was so completely intrathoracic as this one.

That the trauma of the patient's accident was sufficient to cause a partial rupture of the artery there is no doubt. While most of the traumatic cases described were due to penetrating wounds from shot or steel, some were due to

*Dr. Taylor.

simple fractures and not a few to comparatively minor injuries such as a fall on the football field. Simpson (1940-1941) described a number of fatal injuries to the intrathoracic vessels from minor trauma. Nelson, recording his case of innominate aneurysm, mentioned four other cases of aneurysm due to fracture of the clavicle, although no case involved the first part of the left subclavian artery.

During the operation, the question of anastomosis of the arterial ends did arise. In view of the fact that after occlusion of the artery an adequate blood supply to the limb was maintained, it was decided not to take the extra risks of an anastomosis. Although the word "adventurous" is perhaps no longer relevant, one may still ponder on Matas' words, "When operating, the surgeon should not obstinately insist upon re-establishing the continuity of the damaged artery by juggling with complicated, hazardous, uncertain and adventurous methods."

As I have pointed out, apart from Morrision Davies' unpublished case, I know of no direct transpleural approach to the left subclavian artery. In reading the accounts of the operations by other routes, one is impressed by the courage of the various surgeons who undertook them.

By contrast, the operation here described was a relatively simple one. An inspection of the interior of the left thorax reveals at once the whole of the first part of the subclavian artery and its internal mammary and costocervical branches before any dissection is undertaken. Surely this route, alone or combined with a supraclavicular incision in cases where the second and third part of the artery is also involved, is the method of choice. Injury to the pleura is frequently mentioned as a complication of both the anterior and posterior extrapleural approaches. Sometimes it produced undesirable results, sometimes it did not. If the opening is missed or is incompletely sutured, if the anesthetist has not adequate control of the lungs, if air remaining or fluid appearing in the pleural space be not evacuated, then indeed, opening of the pleura may be a disaster. If, however, a deliberate thoracotomy is undertaken, the complication rate will be negligible from this source.

It is undoubtedly presumptuous to argue on the strength of one case, but aneurysm of the first part of the left subclavian artery occurs only rarely in the practice of any surgeon. Diminution in the complications of this condition will occur only when it is recognized that the correct surgical approach to the artery, whether anteriorly or posterolaterally, is by direct thoracotomy.

It might not be out of place at this stage briefly to describe the collateral circulation likely to take place after ligation of the first part of the left subclavian artery (Fig. 5):

1. Between the vertebral artery and the vertebral artery of the opposite side and with muscular branches of the occipital artery
2. Between the inferior thyroid artery and the superior thyroid and other muscular branches from the carotid
3. Between the ascending branches of the transverse cervical and the deep cervical branch of the costocervical, anastomosing with descending branches of the occipital artery

4. Between the descending branch of the transverse cervical, the intercostal branches of the costocervical, the superior thoracic, the long thoracic, and pectoral branches of the thoracic axis anastomosing with the aortic intercostals
5. Between the internal mammary and inferior epigastric and aortic intercostals

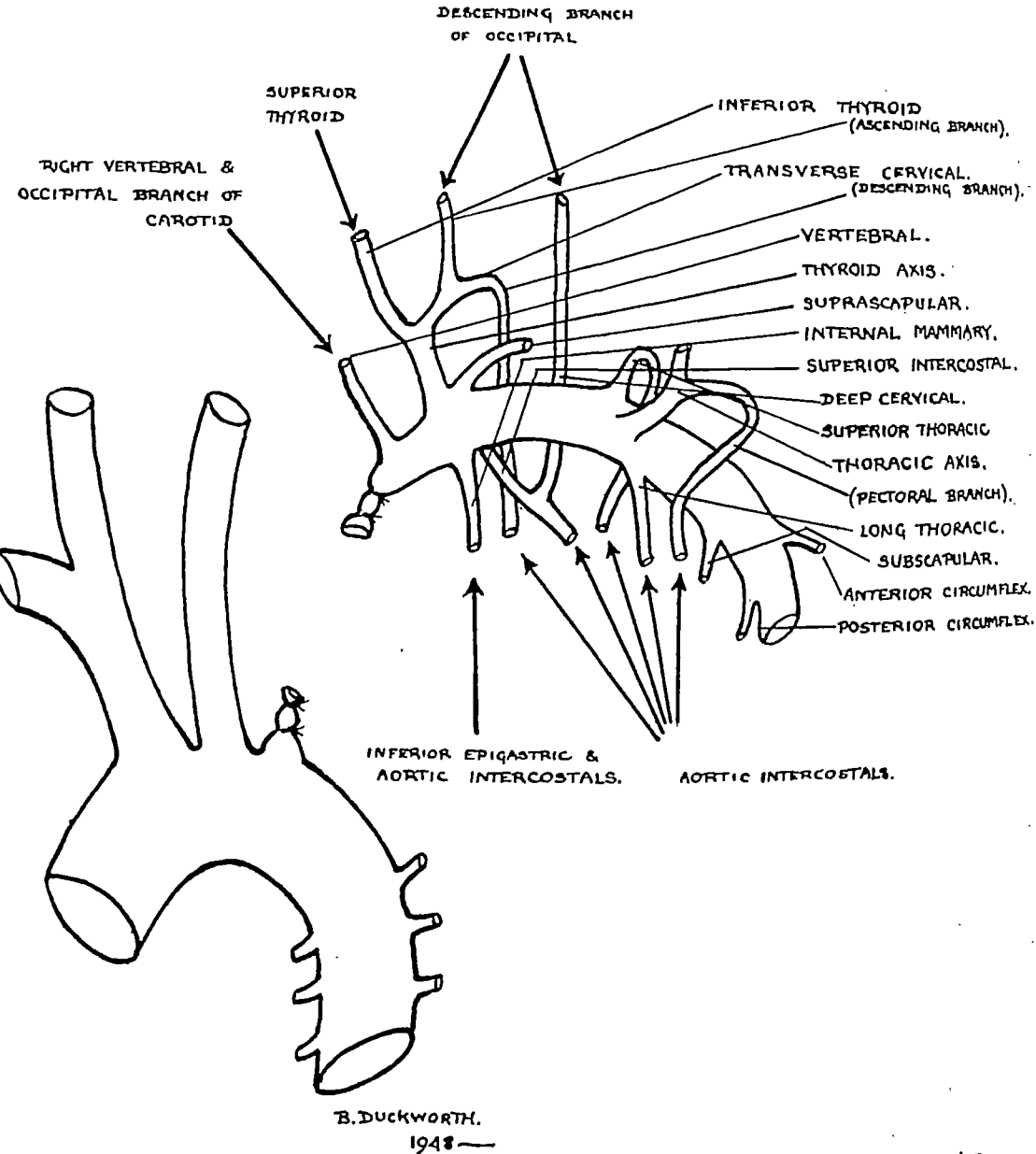


Fig. 5.—Diagram of the anastomosis after ligation of the first part of the left subclavian.

SUMMARY

The literature of aneurysm of the left subclavian artery and ligation of its first part is reviewed. An additional case is described and the diagnosis dis-

cussed. A plea for transpleural exposure of the first part of the left subclavian artery is made. The collateral circulation after ligation of the first part of the left subclavian is briefly mentioned.

In submitting this paper I most gratefully acknowledge my thanks to Miss Duckworth for the drawings, to Mr. H. Morriston Davies for his advice and encouragement, and to Mr. F. Ronald Edwards whose criticism and advice has been invaluable.

REFERENCES

- Adams, H. O.: Arterial Injuries of the Thorax, *J. THORACIC SURG.* 15: 365, 1946.
- Bateh, F.: Richtlinien der Schlagaderchirurgie beim falschen Subclavia-Aneurysma mit Plexuslusion, *Klin. med. Wien.* 2: 364, 1947.
- Edwards, Tudor: War Wounds and Injuries of the Chest, *Brit. J. Surg.* 31: 74, 1943.
- Elkin, D. C.: Treatment of Aneurysms and Arteriovenous Fistulae, *Bull. U. S. Army M. Dept.* 5: 157, 1946.
- Elliott, Ellsworth, Jr.: Treatment of Spontaneous Subclavian Aneurysm, *Tr. Am. Surg. Ass.* 30: 381, 1912.
- Elliott, Ellsworth, Jr.: Subclavian Aneurysm, *Ann. Surg.* 98: 670, 1932.
- Freeman, N. E.: Arterial Repair in Treatment of Aneurysms and Arteriovenous Fistulae; Report of 18 Successful Restorations, *Ann. Surg.* 124: 888, 1946.
- *Halsted, W. S.: Ligation of the Left Subclavian Artery in Its First Portion, *Johns Hopkins Hosp. Rep.* 21: 1, 1920.
- *Henry, A. K.: Method of Ligaturing the First Stage of the Left Subclavian Artery From Behind, *Brit. J. Surg.* 10: 367, 1923.
- Kerly, P.: Intrathoracic Aneurysms, *Brit. J. Radiol.* 12: 158, 1939.
- Kirchner, W. C. G.: Problem of Subclavian Aneurysm: Ligation of Artery at First Portion, *J. Missouri M. A.* 24: 135, 1927.
- Lindskog, G. E., and Howes, E. L.: Cervical Rib Associated With Aneurysm of Subclavian Artery, *Arch. Surg.* 34: 310, 1937.
- *Lee, W. E., Mitchell, C. F., and Peacock, A. B.: Traumatic Aneurysms of the Subclavian Artery, *Ann. Surg.* 100: 87, 1934.
- MacFee, W. F.: Cervical Rib Causing Partial Occlusion and Aneurysm of the Subclavian Artery, *Ann. Surg.* 111: 549, 1940.
- Martin, J. D., Jr.: War Wounds of the Chest, *Surgery* 21: 381, 1947.
- *Matas, R.: A Summary of Personal Experience in the Surgery of the Subclavian Arteries, *Tr. South. S. A.* 39: 213, 1926.
- Miscall, L., and Harrison, A. W.: Thoracic Surgery in a Hospital Center, *Ann. Surg.* 125: 142, 1947.
- Muller, G. P.: Subclavian Aneurysm With Report of a Case, *Ann. Surg.* 101: 568, 1935.
- Nelson, H. P.: Subclavian Aneurysm Following Fracture of the Clavicle, *St. Barth. Hosp. Rep.* 65: 219, 1932.
- Oribasius: Ed. Daremberg. 4: 52. (Quoted by Halsted.)
- Perotti, G.: Aneurisma traumatico dell'arteria ascellare guarito con l'allacciatura della succlavia, *Riforma med.* 61: 232, 1947.
- Rehn, E.: Aneurysm of Subclavian Artery From Gunshot Wounds; Treatment by Transplantation of Vein, *Zentralbl. f. Chir.* 69: 1262, 1942; *Bull. War Med.* 3: 264, 1943 (Abstract).
- Sherrill, J. G.: Report of a Case of Aneurysm With a New Method of Ligation of the Left Subclavian, *Tr. South. Surg. & Gynec. Ass.* 23: 190, 1911.
- Shumacker, H. B., Jr., and Carter, K. L.: Arteriovenous Fistulae and Arterial Aneurysms in Military Personnel, *Surgery* 20: 9, 1946.
- Simpson, Keith: Traction Rupture of the Great Vessels of the Chest Following Injury to the Head, *Guy's Hosp. Rep.* 90: 196, 1940-1941.
- Souchon, E.: Operative Treatment of Aneurysm of the Third Portion of the Subclavian Artery, *Ann. Surg.* 22: 565, 1895.
- Snyder, J. M., and Tropea, F. J., Jr.: War Injuries of the Chest, *Surgery* 22: 780, 1947.
- *Von Haberer: Kriegsaneurysmen, *Arch. f. klin. Chir.* 107: 611-693, 565-567, 1915-1916.
- Wilson, Harwell, and Carr, Disaue: Combined Cervical and Thoracic Approach for Resection of Large Arteriovenous Aneurysm, *Surgery* 24: 342, 1948.
- Wright Smith, R. J.: Left Subclavian Aneurysm, *M. J. Australia* 1: 754, 1927.

*These articles are of particular importance.

THE TREATMENT OF PULMONARY HYDATID DISEASE

M. P. SUSMAN, F.R.C.S.
SYDNEY, AUSTRALIA

IN A most interesting and instructive paper on "Nonmalignant Intrathoracic Lesions Simulating Bronchiogenic Carcinoma" by Brewer, Jones, and Dolley,¹ there appears this statement about the treatment of hydatid cysts of the lung: "Since the best treatment for echinococcus disease is pulmonary resection, if the condition of the patient permits, pulmonary resection is the procedure of choice."

With this advice I heartily disagree, as do most Australian surgeons with experience in the treatment of hydatid cysts. Logan and Nicholson² have written in favor of lobectomy, and the indications for lobectomy have been discussed fully by Barrett.³ In a recent paper⁴ I gave my reasons for opposing many of Barrett's indications. Since then I have had twelve additional cases, and these have served to confirm my advocacy of conservative surgery for most hydatid cysts even when complications have already occurred before operation. Let me quote, from the summary of this paper,⁴ the treatment that I recommended:

When the lung is adherent to the chest wall:

- a. If the cyst is not infected, the contents are removed and the adventitia is closed.
- b. If the cyst is infected, the contents are removed, and the cyst space is drained. Should the pleural cavity be entered during the operation it must be securely closed before opening the adherent cyst.

When the lung is not adherent:

- a. If the cyst is not infected, the pleural cavity is freely opened and protected, the contents of the cyst are removed, the adventitia is closed and sutured to the chest wall, and the pleural cavity is drained, with a water seal, through a stab wound.
- b. If the cyst is infected, the pleural cavity is opened and protected, the contents of the cyst are removed, the cyst space is drained and marsupialized, and the pleural cavity is drained through a stab wound.

Lobectomy may be necessary for the following conditions:

1. Serious hemorrhage from the cyst space during a conservative operation.
2. Residual bronchiectasis, if symptoms warrant it.
3. Empty sac, if hemorrhage or infection warrant it.
4. When the diagnosis is uncertain.
5. Giant cyst.

I have now treated two infected cysts without drainage of the cyst space, just as if they had been uninfected, and in this I have differed from the advice just quoted. This is not to say that my earlier treatment was wrong, but only that it may be made still more simple. The records of these two cases follow.

From Sydney Hospital and the Royal North Shore Hospital.
Received for publication Feb. 15, 1949.

CASE 1.—L. T., a boy aged 13 years, complained of vague discomfort in the right side of the chest in September, 1947. Two months later he had several attacks of hemoptysis at short intervals, but in the following seven months he was apparently quite well. A radiogram taken during this period (April, 1948) showed an opacity in the apex of the right lower lobe (Fig. 1). In June, 1948, after falling from a horse, he had a large hemoptysis and became ill with a productive cough and fever. There were no abnormal signs to be found in the chest, but a radiogram now showed a crescent of air in the opacity (significant of rupture of the cyst into a bronchus) (Fig. 2). The Casoni test gave no reaction, and no hydatid elements were found in the sputum, but a white cell count showed 5 per cent eosinophils.

Under intratracheal anesthesia the thorax was opened through the posterior part of the eighth rib bed. The free pleural cavity was entered and a hard mass in the lung was found adherent to the chest wall just above the incision. The pleural cavity was shut off, a segment of the seventh rib was removed, and an incision through the adherent zone found pus and collapsed hydatid membrane. Most of the pus was aspirated and the remainder was removed along with the membrane. Examination with a spotlight showed several bronchial fistulas, and at the same time I noticed that the cyst space was becoming smaller as the surrounding lung expanded; at this stage the anesthetist aspirated some blood through the Magill tube. The cyst space was dried and powdered with penicillin, and then closed loosely with four interrupted linen thread sutures. The wound was swabbed with 5 per cent formalin and closed with interrupted sutures. Although the cyst was firmly adherent I put a tube through the ninth interspace into the pleural cavity to anticipate possible pleural soiling and to hasten re-expansion of the lung which was free everywhere except at the site of the cyst.

The patient made a quick recovery; for several days he was febrile and he coughed up some bloody sputum. The lung expanded promptly and the intercostal tube was removed on the seventh day. A radiogram taken on the eighteenth day showed an irregular opacity, and he was discharged free of symptoms (Fig. 3).

CASE 2.—J. M., a man aged 20 years, had a brisk hemoptysis after an injury to his chest. In the next seven months he had recurrent hemorrhages and gradually some purulent sputum was brought up between these attacks. His general condition remained good throughout, a point of diagnostic significance. Physical signs were indefinite and bronchoscopy showed nothing abnormal. There was a radiologic opacity in the apex of the left lower lobe (Figs. 4 and 5). At operation I resected a rib too low and the free pleural cavity was opened; there was a firm mass in the lower lobe adherent to the chest wall just above the incision. The pleural cavity was shut off and pus with collapsed membrane were removed from the cyst. The following steps of the operation were as in Case 1. The intercostal tube was removed on the fifth day and a radiogram on the fifteenth day was clear except for lack of translucency at the base (Fig. 6).

These two cases show clearly that after the cyst membrane and its purulent contents have been removed, the residual space and the surrounding lung may be so clean that they can be treated as being uninfected. I had felt that this was so earlier in my experience but had feared to dispense with drainage of the cyst space after pus had been found at operation.

I am now more than ever convinced that the barrier formed by the adventitia makes all the difference between an infected hydatid cyst and an ordinary lung abscess, and that, if there has been no soiling of the lung itself, closure of the cyst space is safe and proper. However, should there be the slightest doubt it is better to drain it.

In the following case I drained an infected adherent cyst without opening the free pleural cavity.



Fig. 1.

Fig. 2.

Fig. 3.

Fig. 1 (Case 1).—Cyst in apex of right lower lobe. (Confirmed by lateral view.)

Fig. 2 (Case 1).—Air space in cyst, due to rupture into bronchus.

Fig. 3 (Case 1).—Radlogram taken eighteen days after operation.



Fig. 4.

Fig. 4 (Case 2).—Cyst in apex of left lower lobe.

Fig. 5.

Fig. 5 (Case 2).—Lateral radiogram, showing cyst in apex of lower lobe.

Fig. 6.

Fig. 6 (Case 2).—Radiogram taken fifteen days after operation. There is incomplete aeration of the lower lobe.

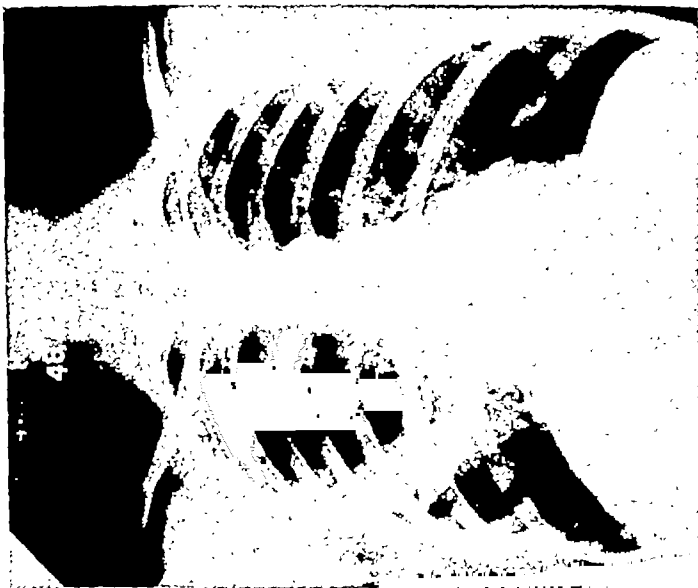


Fig. 9.



Fig. 8.



Fig. 7.

Fig. 7 (Case 3).—Cyst of middle lobe, or adjacent part of upper lobe; it has ruptured into the bronchus.

Fig. 8 (Case 3).—Lateral view corresponding to Fig. 7.

Fig. 9 (Case 3).—Radiogram taken two weeks after operation, showing irregular opacity and cavitation.

CASE 3.—J. C., a boy aged 15 years, gave a history of pleurisy on the right side in January, 1947. He soon recovered without any residual symptoms and remained well for the next eighteen months. Then he had an acute illness with fever and productive cough; some of the sputum was blood stained. Six weeks later he was admitted to the hospital, febrile and anemic with a productive cough and signs of consolidation of the right upper and middle lobes. He improved with chemotherapy and blood transfusion. No hydatid elements were found in the sputum, but the Casoni test was positive. A radiogram showed an opacity with a fluid level in the right middle lobe or adjacent part of the upper lobe (Figs. 7 and 8).

At operation I resected a segment of the fourth rib between the anterior axillary line and the costal cartilage. The cyst was found firmly adherent at this site and it was opened with a small scalpel. Offensive pus and collapsed membrane were removed. Some brisk hemorrhage followed this, but it soon stopped. (Hemorrhage after the opening of a hydatid cyst is sometimes so severe that lobectomy must be performed at once.)

The cavity was several inches in diameter and did not become appreciably smaller under observation; because of this, and because there was a larger bronchial fistula than usual, and because the pus was offensive, I deemed it safer to put in a tube, closing the rest of the wound around it. I passed an bronchoscope at the end of the operation and aspirated some bloody pus from the right bronchus. The patient made a good recovery; the tube was removed on the tenth day and two weeks later a radiogram showed an irregular residual space (Fig. 9). The patient was well and symptomless three months later.

It may be advisable to drain both the cyst space and the pleural cavity, as in the two following cases.

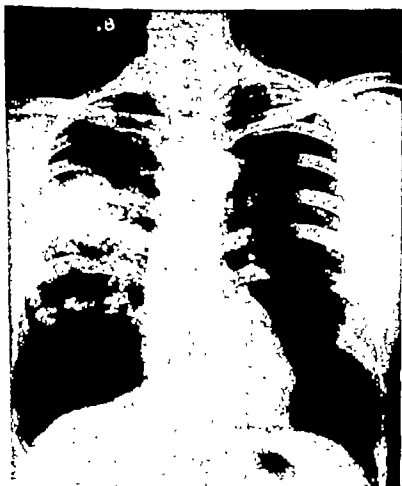


Fig. 10.



Fig. 11.

Fig. 10 (Case 4).—Cyst in right upper-lobe. Although it has ruptured into a bronchus there is no air space or fluid level.

Fig. 11 (Case 4).—Lateral view corresponding to Fig. 10.

CASE 4.—T. F., a man aged 53 years, had a "pneumonia" of the left side eighteen months before his present illness. He was now admitted to the hospital with a suppurating hydatid cyst. There were signs of hyporesonance and diminished air entry over a large part of the right upper lobe, and corresponding to these signs there was a rounded opacity in the radiogram, with surrounding infiltration (Figs. 10 and 11).

In spite of the suppuration his general condition was good. He had about one-half ounce of mucopurulent sputum daily in which no hydatid material could be found. He did not react to the Casoni test but the complement fixation test was positive. Clubbing of the fingers was noted.

At operation I removed a segment of the sixth rib from near the angle to just beyond the scapular line. The lung was seen to be moving under the rib bed, and I deliberately opened the pleura for exploration. I felt a firm mass in the upper lobe adherent farther forward than I had expected, under the fifth rib, near the posterior axillary line. I closed the incision in the sixth rib bed and then opened the adherent cyst from which the crumpled membranes and some debris were removed. Although the cyst space became smaller under observation, I decided to drain it because of some bleeding and because of a fairly large bronchial fistula. In retrospect, I think that it would have been quite safe to have closed the cyst space as in the first three cases. I also put a tube into the pleural cavity for sealed drainage. Although his general state remained good throughout, complete recovery was delayed because of slow re-expansion of the lung. However, within five weeks it had completely re-expanded and he was dismissed well.

CASE 5.—J. R., a girl aged 19 years, had coughed up some "frothy material" three weeks before admission to the hospital. Her condition was poor and she had severe dyspnea; the blood picture showed an eosinophilia of 25 per cent and the hydatid tests were positive. A radiogram showed a large air space in the left upper lobe with an opacity at the bottom (Figs. 12 and 13). During my absence a surgical colleague operated under unsatisfactory conditions, as the anesthetist refused to give the patient a general anesthetic. (From my experience I find that an intratracheal anesthetic is certainly preferable if not absolutely necessary unless one can be certain of finding the lung and cyst adherent.) Under local anesthesia he removed a segment of the fourth rib in the axilla and the free pleural cavity was opened: the cyst was found to be adherent posteriorly. A two-stage operation was decided upon and stitches were passed between the visceral and parietal pleuras to promote further adhesion, and gauze soaked in iodine was left in the wound. The next day the patient was ill and febrile. A radiogram showed that there was a hydropneumothorax below the cyst (Fig. 14). Air was aspirated from the pneumothorax and nine days after the operation a tube was inserted (through the fourth interspace in the axilla) into the pleural cavity. I first saw the patient thirty days after the operation, when she had general constitutional upset, with remittent fever and purulent sputum. At this stage I opened the thorax through the eighth rib bed. The lower lobe was found collapsed against the mediastinum and there was a little fluid in the pleural cavity. The cyst in the upper lobe was adherent, and the membranes and much offensive pus were removed. A tube was put into the cyst space and into the pleural cavity and both were connected to a water-seal drainage system. The wound was closed loosely about the tubes. Her condition was precarious throughout this operation, but she soon showed signs of improvement. The lung gradually became fully aerated and expanded, and a radiogram taken nine months after the last operation showed a well-expanded lung (Fig. 15).

The next two cases show what conservative surgery can do even in the face of the serious complication of rupture of the cyst into the pleural cavity, causing pneumothorax and infection. "Hydatid pneumothorax," though a well-known possibility, is not common if one may judge from the small number of reported cases. It may occur apparently spontaneously or it may fol-

low some trauma such as direct injury, a straining effort, or coughing. Occasionally it is a quiet event with few symptoms at first, but generally, as with other forms of spontaneous pneumothorax, there may be sudden and dramatic

Fig. 12.

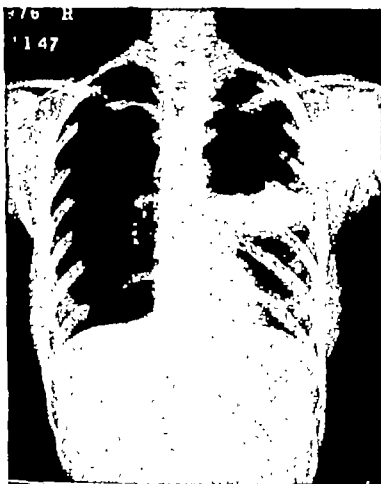


Fig. 13.



Fig. 14.

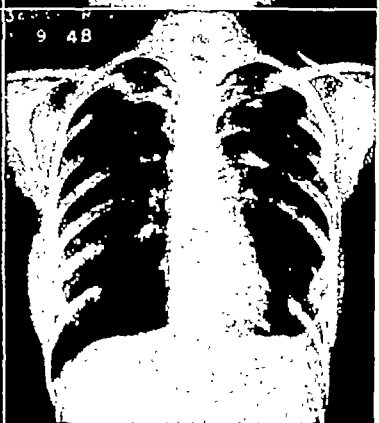


Fig. 15.

Fig. 12 (Case 5).—Hydatid cyst of left upper lobe. The cyst has ruptured into the bronchus and most of the fluid has been coughed up. The collapsed hydatid membranes lie at the bottom of the air-filled cyst.

Fig. 13 (Case 5).—Lateral view corresponding to Fig. 12.

Fig. 14 (Case 5).—There is now a hydropneumothorax below the cyst.

Fig. 15 (Case 5).—Radiogram taken nine months after last operation. The lung has completely re-expanded.

symptoms which may be more overwhelming than usual because of the added symptoms of anaphylaxis. If operation is delayed, pleural infection is almost inevitable, and still later there may be secondary hydatid cysts in the pleural cavity. It is clear that prompt operation is essential as soon as the pneumothorax occurs, both to save life and to avoid further complications; the ruptured membranes and all fluid and debris must be removed, any obvious bronchial fistulas should be closed, and the pleural cavity should be drained by a water seal. In the two cases here reported pyopneumothorax was already present before the patients came into my ken.

CASE 6.—A man, aged 56 years, coughed up some fluid and soon afterward had a spontaneous pneumothorax followed by fever and dyspnea and failure of the lung to re-expand. A radiogram showed a collapsed and airless lung with a complete pneumothorax. Thoracoscopy three months later showed that the collapsed lung was covered with a layer of white fibrin. At this stage he had one-half cup of yellow sputum daily; no hydatid elements were



Fig. 16 (Case 6).—Radlogram taken one and one-half years after operation.

found in it. The Casoni test was negative, but the complement fixation was positive. Bronchoscopy and bronchography gave no positive information. It was decided that exploratory thoracotomy was necessary both for diagnosis and treatment. I opened the chest wall through the fifth rib bed and noted thickening of the parietal pleura. The whole lung was collapsed and covered with thick fibrin. I set out to decorticate the lung and a good "peel" was found in the costovertebral groove, but further forward the fibrin layer was closely attached to the lung and the decortication was incomplete. Just above the diaphragm I came upon a deep excavation in the lung with several bronchial fistulas, and after a little more manipulation I found collapsed hydatid membrane lying between the diaphragm and the base of the lung. The excavation in the lung was evidently the original site of the cyst before its rupture into the pleural cavity.

I decorticated the lung as thoroughly as possible and, when the bronchial fistulas had been closed, it aerated slightly when the anesthetist raised the pressure. Two drainage tubes were left in the pleural cavity, one in front and one behind; they were both connected to a

water sent, and the main wound was closed. Later, suction was applied to the drainage tubes. The lung re-expanded slowly after prolonged drainage and Fig. 16 is the radiogram taken one and one-half years later; although the general condition of the patient improved he had repeated small hemoptyses in the following year. Bronchoscopy and bronchography showed no abnormality. It is now over two years since operation, and he is well except for moderate dyspnea on exertion and occasional staining of the sputum.

CASE 7.—II. L., a woman aged 45 years, had a "pneumonia" in December, 1947. After recovery from this, a radiogram showed a hydatid cyst of the left lung. Just before admission to the hospital the cyst ruptured into the pleural cavity and bronchus. Signs of pneumothorax were found, and soon there was evidence of sepsis in the lung and the pleural cavity. A radiogram showed the pneumothorax with a fluid level, and just above and medial to it part of the lung was visible in which there was a small cavity with a fluid level; the mediastinum was displaced to the right (Fig. 17). She reacted to the Casani and complement



Fig. 17.

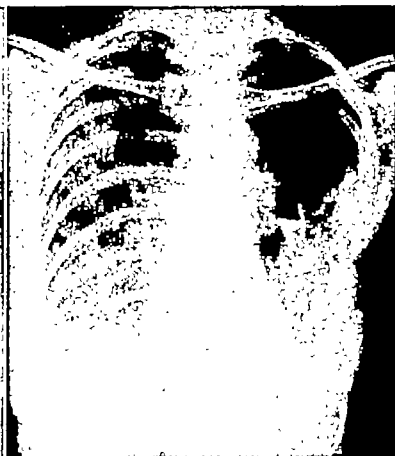


Fig. 18.

Fig. 17 (Case 7).—Large pyopneumothorax, with displacement of heart to the right. Just above and medial to the fluid level in the pleural space there can be seen, in the collapsed lung, the ruptured hydatid cyst with a fluid level.

Fig. 18 (Case 7).—Radiogram taken seven months after the final operation (for plastic closure of the residual empyema cavity).

fixation tests, but no hydatid material was found in the sputum. The first operation was simple closed drainage of the empyema through a small rib resection (sixth) in the mid-axillary line. This gave prompt relief and within two days the upper lobe had become fully expanded; the lower half of the lung field remained opaque and the mediastinum became central. She still had some purulent sputum, but it was much less than on admission. One month after this operation thoracotomy was done; six inches of the eighth rib were removed and an empyema cavity was opened, containing a little pus and crumpled hydatid membrane. The upper lobe was firmly adherent to the chest wall, the lower lobe moved on respiration, and two bronchial fistulas were seen; these were closed with several silk sutures. The main wound was closed except posteriorly where a tube was put in for drainage. Two months later her general condition was good, but there was still a chronic empyema space and one or both of the bronchopleural fistulas had reopened. Another operation was performed for un-

roofing of the empyema cavity and the filling of it with a large flap from the latissimus dorsi muscle and two intercostal muscles. This operation served to close the defect and she was soon well except for pain in the course of the lower intercostal nerves. Fig. 18 is the radiogram taken seven months later.

CONCLUSIONS

Most hydatid cysts of the lung can be adequately treated by conservative surgery, consisting of removal of the cyst membranes and fluid and drainage of the pleural cavity or of the cyst space or of both, according to the conditions found. Even when complications have already occurred, conservative surgery usually suffices. Lobectomy is needed for a small number of hydatid cysts.

Editor's Note: The illustrations present no postoperative bronchograms. When the author was questioned about his failure to include such bronchograms, he stated that he had not made them routinely because all but two of the patients recovered quickly and apparently completely. In the two cases in which the recovery was not so rapid, bronchograms were made and were found normal.

REFERENCES

1. Brewer, Lyman, A., Jones, Wilfred M. G., and Dolley, Frank S.: Nonmalignant Intrathoracic Lesions Simulating Bronchiogenic Carcinoma, *J. THORACIC SURG.* 17: 439, 1948.
2. Logan, A., and Nicholson, H.: Hydatid Disease of the Lung, *Thorax* 3: 1, 1948.
3. Barrett, N. R.: The Treatment of Pulmonary Hydatid Disease, *Thorax* 2: 21, 1947.
4. Susman, M. P.: The Treatment of Pulmonary Hydatid Disease, *Thorax* 3: 71, 1948.

BRONCHIECTASIS ASSOCIATED WITH ANOMALY OF THE RIGHT PULMONARY VEIN AND RIGHT DIAPHRAGM

REPORT OF A CASE

EMERSON H. DRAKE, M.D.,* PORTLAND, ME., AND JOSEPH P. LYNCH, M.D.,†
BOSTON, MASS.

THE increased frequency with which resection of lobes of the lung is performed at present must mean that congenital anomalies within the thorax are being more frequently encountered. However, there are few reports of such anomalies in the medical literature. This is unfortunate, since a clear knowledge of the possible anomalies and their significance is important to the surgeon, both in understanding the pathogenesis of disease and, technically, for the safety of the patient. The case presented in this report illustrates a combination of anomalies, one of which was probably responsible for the patient's disease process. The other made surgery technically more difficult.

It is of interest to note that there is an increased incidence of pathologic processes in lungs containing congenital anomalies of one type or another. Gruenfield and Gray¹ have discussed the common congenital malformations of the lungs, pointing out the increased incidence of pulmonary tuberculosis and bronchiectasis in the azygos lobe. Womack and Graham² have analyzed the incidence of bronchiogenic carcinoma in lungs with congenital abnormalities. Adams and Churchill³ have likewise considered the more frequent occurrence of bronchiectasis in patients with situs inversus. The present case does not fit into the classification of the usual abnormalities, and since it offered a puzzling picture preoperatively as well as at the time of operation, it seems worthy of presentation.

CASE REPORT

J. L. (R-13879), a 24-year-old man, an accountant, was admitted to the Veterans Administration Hospital, West Roxbury, Mass., on July 28, 1947, complaining of a cough of five years' duration. The cough was most marked in the morning and was productive each day of one-half cupful of yellowish, nonfoul but occasionally blood-flecked sputum. Two years prior to admission, the patient had had a hemoptysis of one cupful of bright red blood. There was also a history of exertional dyspnea after climbing one flight of stairs.

Physical examination was noncontributory except for the presence of diminished breath sounds and fine crepitant rales at the right base anteriorly and posteriorly. The left lung was normal.

X-ray examination of the chest (Fig. 1) showed that the heart was displaced to the right but was normal in rotation, and with the aortic arch in its normal position. In the lateral view, a linear shadow was seen extending posteriorly from the apex of the diaphragm to the posterior part of the chest wall. These roentgenograms were interpreted as demon-

From the Surgical Service, Veterans Administration Hospital, West Roxbury, Mass.

Published with the permission of the Chief Medical Director, Department of Medicine and Surgery, Veterans Administration, who assumes no responsibility for the opinions expressed or conclusions drawn by the authors.

Received for publication Feb. 28, 1949.

*Former Assistant Chief, Surgical Service, Veterans Administration Hospital, West Roxbury, Mass.

†Attending Thoracic Surgeon, Veterans Administration Hospital, West Roxbury, Mass.

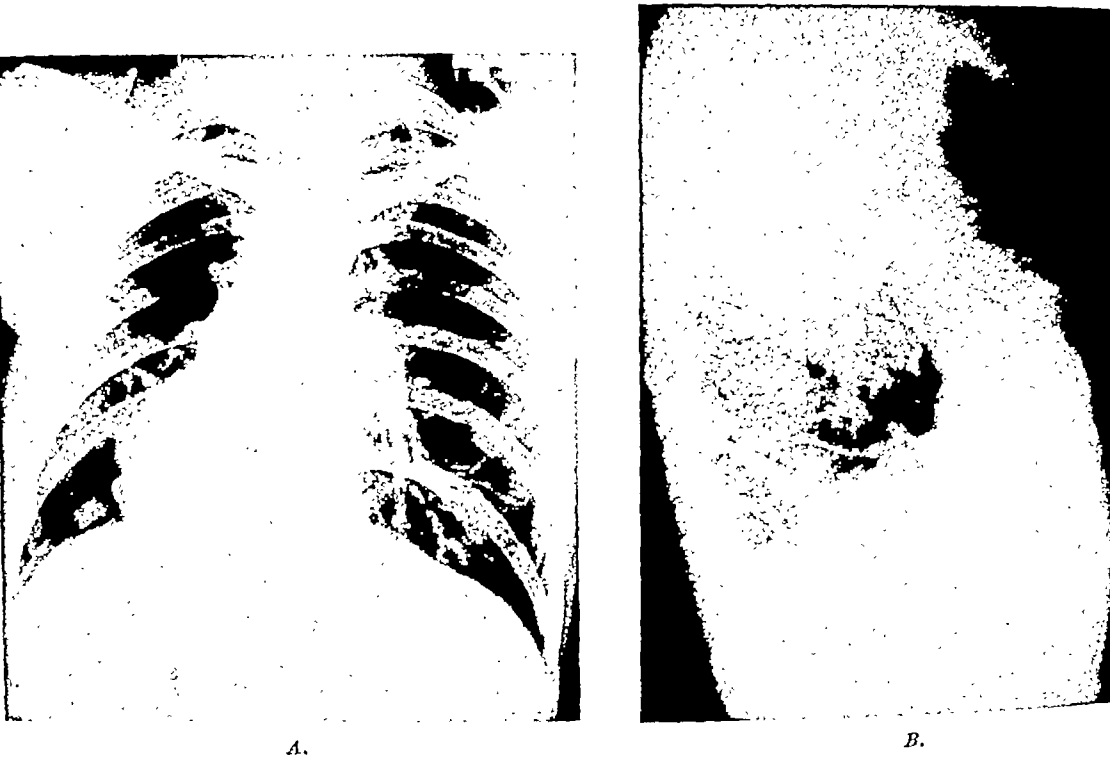


Fig. 1.—*A*, Posteroanterior roentgenogram of chest. *B*, Right lateral roentgenogram of chest on admission.



Fig. 2.—Right lateral bronchogram of chest.

strating a partially atelectatic lower lobe with increased aeration of the middle lobe. The previously mentioned linear shadow was interpreted as the fissure between the middle and lower lobe.

At bronchoscopy, the mucous membrane of the right lower lobe was slightly redder than normal but otherwise examination was not remarkable. A bronchogram demonstrated a normal left lung. On the right side there was cylindrical bronchiectasis of the anterior basal division of the right lower lobe. The other basal segments appeared decreased in size with crowding of the bronchi as seen in the lateral view (Fig. 2). It was felt that the symptoms could be accounted for by pathology in the right lower lobe, and operation was advised.

At the time of thoracotomy, the right lower lobe was found to be divided into two portions by an extra leaf of the diaphragm which arose from the dome of the normal right diaphragm and extended laterally and posteriorly to join the chest wall two inches above the normal diaphragmatic insertion. The two portions of the lower lobe were connected posteromedially where this accessory leaf did not reach the chest wall. Through this connecting isthmus of the lung, the vascular and bronchial supply of the trapped segment passed. Beneath this "backpocket" segment was a normally attached, complete diaphragm (Fig. 3).

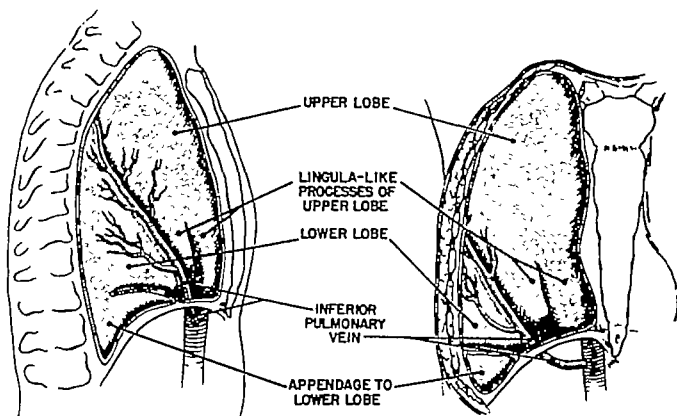


Fig. 3.—Lateral and anterior illustration of venous and diaphragmatic anomaly.

There was no true fissure between the middle and upper lobes. What should have been the middle lobe consisted of two lingular processes extending over the right border of the heart, which lay to the right of its usual position.

In the fissure between this upper and the anomalous lower lobe was discovered a large vein which proved to be the inferior pulmonary vein (Fig. 3). It extended downward and anteriorly through the fissure, receiving branches from the lower and upper lobes, to penetrate the diaphragm at its mid-point just anterior to the junction of the normal and anomalous leaves. On opening the diaphragm this vein was found to run medially along its inferior

fifth rib in the midaxillary line to the iliac crest and from two inches lateral to the vertebral column posteriorly to the umbilicus anteriorly. The skin over and immediately surrounding the tumor was of a dull, reddish-purple color and was shiny and indurated; several areas of ulceration were present. The surface of the tumor was very irregular and loculated due to the presence of multiple cystic areas of varying sizes. There was considerable tenderness to palpation and the tumor was fixed to the underlying structures. Several of the cystic areas were aspirated. Three hundred cubic centimeters of red-tinged, serous fluid were obtained, thus relieving the pain slightly.

X-ray and fluoroscopic examination revealed a large area of slightly increased density over the lower left part of the chest extending from the fifth rib anteriorly to the crest of the ilium, and to the mid-portion of the abdomen. The ninth, tenth, eleventh, and twelfth ribs were involved; no excursion of the diaphragm could be made out. An upper gastrointestinal x-ray examination revealed the stomach and small bowel to be displaced markedly to the right.

It was decided an attempt at removal should be made despite the patient's poor general condition. The approach decided upon was transthoracic, in an attempt to get behind the tumor and enucleate it in toto.

The patient was admitted to Mt. Carmel Hospital, Columbus, Ohio, on Aug. 29, 1948.

Laboratory Work.—Red blood count was 3,600,000 and white blood count, 8,000. Aug. 30, 1948, urinalysis showed 250 mg. per cent of albumin, plus fine granular and cylindrical casts; 2 plus bacteria, 2 plus mucus. Aug. 31, 1948, albumin was 400 mg. per cent, cylindrical casts, 3 plus. Total proteins on Aug. 30, 1948, were 5.8 mg. with 4.1 to 1.7 albumin-globulin ratio. Serology was negative.

Operation.—On Aug. 31, 1948, the patient was operated upon under endotracheal anesthesia and the tumor was removed.

A left posterolateral thoracotomy incision was made above the tumor, through the bed of the eighth rib which was resected subperiosteally. The diaphragm was incised; the peritoneal cavity was entered and by manual palpation the tumor was found to extend beyond the umbilicus. A second incision was made from the anterior end of the transthoracic incision to the crest of the ilium and a skin flap definitely free of tumor tissue was mobilized by sharp dissection. The skin was ulcerated and markedly indurated due to the previous x-ray therapy and probable local pressure necrosis. The descending colon and stomach were separated from the tumor from below upward by sharp and blunt dissection. The tumor was rotated laterally from its medial surface and finally removed in that manner with blunt and sharp dissection. Due to the involvement of the chest wall, the anterior and lateral portions of ribs twelve, eleven, ten, and nine, together with their intercostal bundles, were removed.

After removal of the tumor mass, the peritoneal cavity was closed without difficulty. The diaphragm was then repaired and a new attachment was made posteriorly to the stumps of ribs nine and eight, and anteriorly to the costochondral junction at about the level of rib five, bringing about a complete closure of the diaphragm at a considerably higher level than normal, the pleural cavity having been drained with two No. 30 catheters attached to a water trap.

A cigarette drain was left in the angle near the crest of the ilium. Approximately 3,000 c.c. of citrated blood were given during the procedure per ankle cannula although very little bleeding occurred. A postoperative bronchoscopy was done. The patient was returned to his room in good condition.

Because of the extensive tissue changes in the skin, débridement had to be carried out at a later date and when an attempt at healing by granulation was unsuccessful, the area was covered with a split-thickness skin graft.¹⁸ The general condition of the patient was good, the skin graft being completed.

Pathology.—The surgical specimen consisted of a massive tumor weighing 3¼ kg. (7½ pounds). It measured 20 by 15 by 15 cm., and was lobulated and very irregular in shape. Attached to the upper portion were four pieces of rib.

GIANT-CELL TUMOR OF RIBS

A CASE REPORT AND SURVEY OF LITERATURE

MAURICE G. BUCKLES, M.D.,* AND EDWARD C. LAWLESS, M.D.†
COLUMBUS, OHIO

THE exact number of cases of primary giant-cell tumors of the ribs is difficult to determine due to the lack of adequate pathologic descriptions. In previous surveys by Hedblom¹ in 1933, Samson and Haight² in 1935, Sommer and Major³ in 1942, Dorner and Marcy⁴ in 1948, there have been nine cases of giant-cell tumors of the thoracic cage reported dating from Fort's⁵ case in 1914 to Janes's⁶ case in 1939. Of these only eight have been reported in literature. Neil's, as described by Samson and Haight,² was presented by personal communication. Codman described a specimen of giant-cell tumor of costal origin at the Warren Museum, Harvard Medical School. Christensen likewise described a case of costal giant-cell tumor, but all records have been lost.

Giant-cell tumors of the thoracic cage have no great significance, per se; however, due to their infrequency as compared to the incidence of giant-cell tumors of other parts of the bony framework, the subject becomes of medical interest.

As well as can be determined from literature, this case to be presented is the largest giant-cell tumor of the thoracic cage, which may well be due to its duration of approximately eight years. A biopsy made six years previously revealed it to be a giant-cell tumor.

CASE REPORT

The patient, a 49-year-old, white man, a confectioner, was first seen on Aug. 28, 1946, with the complaints of (1) a "large tumor" on the left side of the chest, (2) pain over the region of the tumor which was not relieved by the usual analgesics, (3) weakness, (4) slight fever, and (5) general lack of well-being.

Present Illness.—The patient stated that in December, 1940, he noticed the appearance of a tumor on the left lower part of the chest. The tumor made its appearance about two months after an injury due to a fall down a flight of stairs. The tumor gradually enlarged and caused considerable pain and discomfort. After the tumor had been present for eighteen months the patient was admitted to a hospital where removal was attempted in June, 1942. Removal at that time was abandoned due to excessive hemorrhage and only a biopsy was obtained. The diagnosis by the pathologist at that time was benign giant-cell tumor of bone.

The mass continued to grow slowly and became more painful. In 1943, the patient received deep x-ray therapy with a total of 2,400 roentgen units in nine treatments given. Again in 1948 he received 3,060 roentgen units in seventeen treatments given over a period of six months. There was no regression in size of the tumor and no relief of pain. The severity of the pain had gradually increased.

Physical examination revealed a well-developed, well-nourished, white man, 49 years of age. The temperature was 101° F., pulse 104, respiration 24, the blood pressure was 180 systolic and 110 diastolic. The only finding of note was the tumor, which was a large, hard, protruding mass, located on the left lower part of the chest and extending from the

Received for publication March 8, 1949.

*Assistant Professor of Thoracic Surgery, Ohio State University, and Attending Thoracic Surgeon, Franklin County Tuberculosis Hospital.

†Former Assistant in Surgery, Ohio State University.

AUTHOR	AGE (YR.)	SEX AND RACE	SITE	SIZE	SYMPTOMS	DURATION	TREATMENT	RESULTS	HISTOLOGIC DIAGNOSIS	TRAU-MA	REMARKS
Fortis	11	M, W	1st rib, clavicle ant.	5 x 3 x 3 cm.	Tumor	8 mo.	Excision post. of 1st rib, clavicle sternum, 1914	No deformity, well 17 yr. later	Benign giant-cell tumor	No	
Gazewood ¹	14	F, W	Left 12th rib, post.	5 x 3 x 3 cm.	Pain, tumor, scoliosis	1 yr.	Excised following by radiation therapy, 1921	Well 13 yr. later	Benign giant-cell tumor	Yes	
Peirces	17	M, W	Left 10th rib, post.	6 inch	Pain, tumor, loss of weight (20 lbs.), dry cough	9 mo.	X-ray with excision of ribs 8 to 12, left, 1930	Died 40 hr. postoperative	Chondro-osteogenic (benign) sarcoma in giant-cell tumor of rib infiltrating	Yes	Only reported case with malignant changes in giant-cell tumor of rib
Hedblom ¹	38	F, W	Right 8th rib, post.	7 x 2 1/4 cm.	Pain, tumor	3 yr.	Excision, 1922	Well 11 yr. later	Benign giant-cell tumor	Yes	Injury 12 yr. before tumor evident; intercostal no tony (8th) 1919 gave relief
Holland ⁹	48	M, W	3th rib, costochondral junction	4 x 3 x 3 cm.	Tumor	6 mo.	Excision, 1923	Well 11 yr. later	Benign giant-cell tumor	No	
Neill ²	9	M, W	Left 4th at angle	Small	Pain	2 mo.	Biopsy, re-dium, 1923	Well 11 yr. later	Benign giant-cell tumor	Yes	
Samson and Haight ²	31	F, W	Rt. 2nd rib, costochondral junction	3.5 x 2 x 2 cm.	Tumor	2 yr.	Excision, 1935	Well 5 yr. later	Benign giant-cell tumor	No	
Iliff ¹⁰	15	F, W	Left 5th rib at angle	3.5 x 3 x 3 cm.	Pain, tumor	6 mo.	Biopsy, 1935; x-ray, 1935	1 yr. and 10 mo. later tumor smaller	Benign giant-cell tumor	Yes	
Jones ⁶	49	M, W	Rt. 7th rib, post.	8 x 5 cm.	Pain	10 mo.	Excision, 1936	Died 2 1/4 yr. later of bronchial ca.	Benign giant-cell tumor	No	
Authors, ¹ case	55	M, W	Left 11th rib with thoracic wall invasion and abdominal extension	20 x 15 x 15 cm.	Tumor, pain, ulceration	8 yr.	Biopsy, 1942; x-ray, 1943; excision, 1948	Well 5 mo. later	Benign giant-cell tumor	Yes	Nine x-ray treatments, 1943; 17 x-ray treatments, 1948; no apparent regression

The tumor was firm in consistency in most places but one large egg-shaped mass on the outer surface measuring 15 cm. in diameter was cystic to palpation. The color of the tumor was mottled yellow, red, and light tan. In some areas there was blackish discoloration due to old hemorrhage. The solid portion on cutting was granular and the color of putty. On the inner surface numerous small cystic areas were palpated. On cutting, the cystic areas were filled with thin, watery, reddish discolored fluid and the walls were covered with multiple loculated areas, some of which were filled with fibrin and blood pigment.

Microscopic examination of sections taken from the tumor revealed a pleomorphic picture. The cells were spindle shaped and fusiform and there were numerous multinucleated giant cells. Diffusely scattered throughout were numerous fibroblasts and large areas of hyalinized fibrous connective tissue. There was a thin hyalinized fibrous connective tissue capsule which was well delineated from the tumor. In the more active areas there were numerous small blood vessels and nodules of siderofibrotic character. There was no evidence of malignancy. The final diagnosis was giant-cell tumor with degeneration and fibrosis.

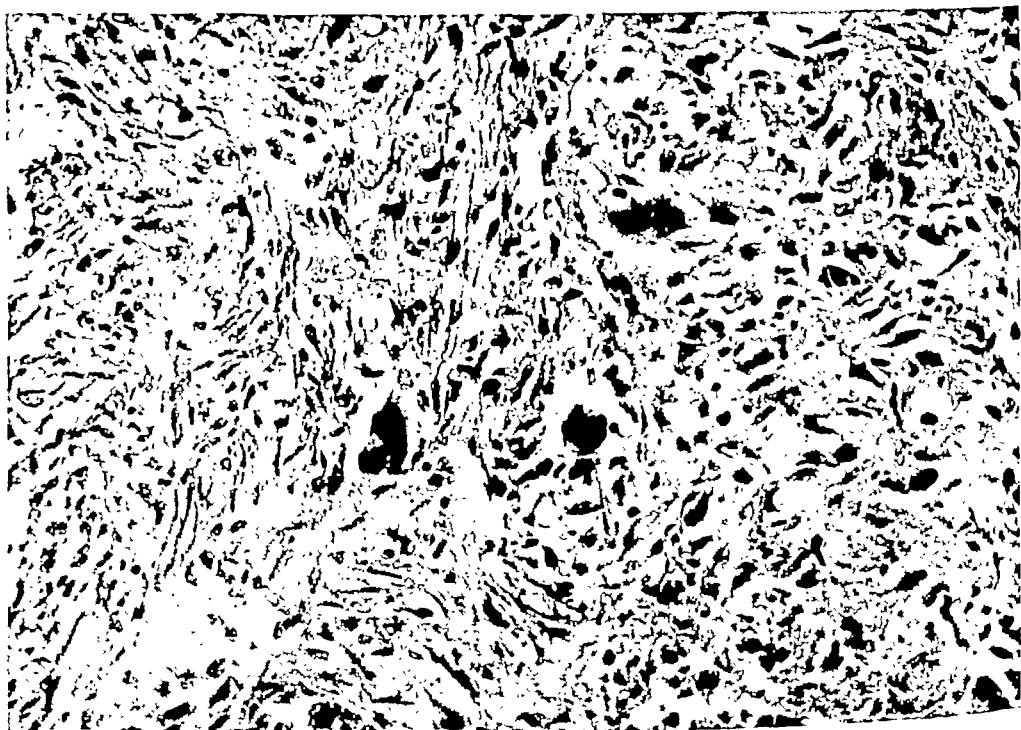


Fig. 1.—Photomicrograph of removed tumor.

DISCUSSION

A summary of the collected cases of giant-cell tumors of ribs is presented in Table I.

The usual clinical history given by patients with giant-cell tumors of costal origin is similar to that obtained in giant-cell tumors in other locations; namely, the sequence of trauma, pain, and tumor extending over a period of from two to sixteen months. Trauma has been fairly well established as a definite etiological factor in the relation of onset of the disease, although in this series of ten cases, trauma was denied in four instances.

5. Fort, R. E.: Excision of the Clavicle and First Rib for Malignant Disease, Surg., Gynec. & Obst. 18: 696, 1914.
6. Jones, R. M.: Primary Tumors of Ribs, J. THORACIC SURG. 9: 145-150, 1930.
7. Gatewood: Sarcoma of Rib, S. Clin. North America 2: 811, 1922.
8. Peirce, C. B.: Giant Cell Tumor of Bone, Am. J. Roentgenol. 28: 167, 1932.
9. Holland, C. T.: The Benign Giant Cell Tumor of Bone, Brit. J. Radiol. 37: 227, 1934.
10. Hilt, L. M.: Benign Giant Cell Tumor of a Rib, Am. J. Roentgenol. 37: 663, 1937.
11. Harper, F. R.: Benign Chondroma of the Ribs, J. THORACIC SURG. 9: 132-144, 1930.
12. Geschickter, C. F., and Copeland, M. M.: Tumors of Bone, Am. J. Cancer 26: 155, 1930.
13. Meyerdling, H. W.: Benign and Malignant Giant Cell Tumors of Bone, J. A. M. A. 117: 1849, 1941.
14. Geschickter, C. F., and Copeland, M. M.: Recurrent and So-called Metastatic Giant Cell Tumor, Arch. Surg. 20: 713, 1930.
15. Kinsella, T. J.: Thoracic Tumors, Clinica 3: 1246, 1915.
16. Hedblom, C. A.: Tumors of the Bony Chest Wall, Arch. Surg. 3: 556, 1921.
17. Zininger, M. M.: Tumors of the Wall of the Thorax, Ann. Surg. 92: 1043, 1930.
18. Maier, H. C.: Surgical Management of Large Defects of Thoracic Wall, Surgery 22: 169, 1947.

The rarity of giant-cell tumors of costal origin, as suggested by Geschickter and Copeland,¹² is perhaps due to the extremely small and relatively inactive epiphyses of the ribs. In this series, four of the tumors were posterior, two were at the angle, two were at the costochondral junction, and two involved an entire rib.

The size of the tumor does not seem to be in proportion to the duration of symptoms, although in the authors' case definitive treatment would have been considerably easier had surgery been carried out earlier.

Due to the inadequate series, it is extremely difficult to draw any definite conclusions as to the proper treatment. The duration between onset and treatment of the tumor does not appear to influence the success of the definitive treatment unless, as in Peirce's⁸ case, malignancy is involved. Direct excision was used in five cases, irradiation only in two cases, excision followed by irradiation in one case, and irradiation followed by excision in one case.

According to Meyerding,¹³ radiologists have discovered that benign and low-grade malignant giant-cell tumors are radiosensitive. While x-ray therapy may be effective in some cases it apparently had no effect on the authors' case, either in controlling pain or diminishing the size of the tumor. Although giant-cell tumors are typically benign, recurrence after surgical excision is not rare and scattered reports in the literature are continually suggesting the possibility of metastases.¹³ This fact has not been borne out in giant-cell tumors of costal origin but as the series is small, no definite conclusions can be made. Invasion of soft parts is not uncommon and was demonstrated in four of the cases: Fort,⁵ Peirce,⁸ Hedblom,¹ and the authors' case. The invasive power of giant-cell tumors may be an explanation of the occasional recurrence. However, in spite of possible malignant changes that may occur or the possibility of recurrence, the prognosis following adequate treatment has been excellent.

SUMMARY

1. A case of giant-cell tumor of costal origin is presented. Its size and duration significantly demonstrate the benignity of these tumors. That these tumors are uncommon is evidenced by only nine other authentic cases being found in the literature.

2. Treatment is variable; excision, irradiation, or combination of these methods give apparently satisfactory end results.

3. It is the belief of the authors that the treatment of choice is early surgical excision, while the tumors are still small, and before they invade the soft parts. In so doing, recurrence and malignant changes will be largely avoided.

4. Providing the therapy has been adequate and malignant changes have not yet taken place, the prognosis is excellent.

REFERENCES

1. Hedblom, Carl A.: Tumors of the Bony Chest Wall, *Ann. Surg.* 98: 528, 1933.
2. Samson, P. C., and Haight, C.: Giant Cell Tumors of Ribs, *J. A. M. A.* 105: 1020, 1935.
3. Sommer, G. N. J., Jr., and Major, R. C.: Neoplasma of the Bony Thoracic Wall, *Ann. Surg.* 115: 51-83, 1942.
4. Dorner, R. A., and Marcy, D. S.: Primary Rib Tumors, *J. THORACIC SURG.* 17: 690-704, 1948.

The patient had always been well with the exception of "arthritis" in several joints which had been troublesome for about eleven years. Recently she had noticed a slight increase of dyspnea on exertion.

Examination revealed a well-developed and well-nourished white woman who appeared to be about the stated age. There was a mass about 7 cm. in diameter which was firmly fixed to the anterosuperior portion of the sternum and extended down to the third interspace. It was fixed to the bone beneath but not attached to the underlying skin. It was smooth, hard, and somewhat tender. The heart and lungs were essentially normal. The blood pressure was 190/90.

Urine examination revealed a specific gravity of 1.220, and a trace of protein. The hemoglobin was 14.0 per cent, and the white blood count 8,250 with 69 per cent polymorphonuclears. The blood glucose was 102, total protein 7.1, blood calcium 9.8, and blood urea nitrogen 9. The globulin was not increased. Inorganic phosphorus was 3.2; alkaline phosphatase was 27.1. The Hinton test was negative.



Fig. 1 (Case 1).—The tumor is seen bulging into the soft tissues over the manubrium.

X-ray examination of the skull was negative. The entire spine and pelvis showed marked changes in several of the dorsal and lumbar vertebrae and all the pelvic bones, characteristic of advanced Paget's disease. There was a collapse of the body of the fourth lumbar vertebrae with slight expansion in both the anteroposterior and lateral directions. The cervical spine showed moderate hypertrophic changes but no other bony pathology. Both lower extremities also disclosed marked involvement of the femurs and upper articular ends of the tibiae due to Paget's disease. The right tibia also showed a slight thickening of the cortex on the lateral aspect of the mid-shaft. Films of the sterno-clavicular region revealed a marked rarefaction and expansion of the manubrium of the sternum. The anterior portion was irregular and showed destruction with invasion or infiltration of the underlying soft tissues. The impression was that of advanced Paget's disease, involving several bones. There was a destructive malignant bone lesion of the manubrium and sternum, probably sarcoma on the basis of associated Paget's disease.

On Feb. 7, 1947, a biopsy of the tumor was taken, and the pathologic examination revealed osteogenic sarcoma of the sternum.

Operation was performed on March 11, 1947. Endotracheal cyclopropane anesthesia was used.

THE USE OF TANTALUM PLATE WHEN RESECTING LARGE AREAS OF THE CHEST WALL

J. MURRAY BEARDSLEY, M.D.*
PROVIDENCE, R. I.

THE resection of large areas of the chest wall which are the seat of malignant disease has a much wider application at the present time than was heretofore possible. This type of operation, which is more frequently associated with pulmonary neoplasms and which may also include the removal of one lobe or the entire lung, exemplifies the present status of excision surgery in this region.

Cancer of the lung with extension to the pleura and ribs has in the past been one of the criteria of inoperability. This concept is being revised in the light of our present knowledge that peripheral lesions have a tendency to local invasion rather than to early spread to the mediastinum as is the case with those more centrally located.

When the disease is so situated that the adjacent chest wall may be removed with the tumor, hope is offered to a relatively large group of patients whose plight was formerly considered hopeless. An additional favorable factor lies in the fact that one of the major lobes can more frequently be preserved than is the case with lesions of the main-stem bronchi.

The excision of a large segment of chest wall gives rise to certain problems that must be considered and dealt with. Paradoxical respiration must be guarded against when there is a mobile mediastinum, and when dealing with large defects this cannot always be satisfactorily controlled by external appliances or pressure dressings. If the hazards of the postoperative period can be surmounted, there nevertheless remains a flabby chest wall which is subject to motion with each respiratory effort.

When, in addition to the bony support, all of the overlying soft tissues must be removed, as was true in Case 1, satisfactory coverage must also be provided.

The following cases illustrate methods of overcoming these problems. In addition to eliminating the immediate postoperative dangers of a weakened chest wall, permanency is created in the defective area.

CASE REPORTS

CASE 1.—C. T., a 63-year-old white woman, was admitted to Rhode Island Hospital on Feb. 5, 1947, complaining of a gradually enlarging mass in the upper part of the sternum. She first noticed discomfort and a small nodule in this region in June, 1946. She consulted her physician who advised observation and that she return if there was any enlargement of the nodule. The mass remained reasonably static until early in December, when it appeared to increase in size rather rapidly, but she failed to consult her physician again until two weeks prior to admission to the hospital.

From the surgical service of the Rhode Island Hospital.
Received for publication Jan. 27, 1949.

*Visiting surgeon and surgeon to the Thoracic Clinic. Rhode Island Hospital. Surgeon-In-Chief, Charles V. Chapin (Providence City) Hospital.

scattered hemorrhagic areas in some of the gelatinous material. This tumor extended into the sternoclavicular joints, but did not involve the clavicles proper. It occupied two-thirds of the manubrium. The gross diagnosis was osteogenic sarcoma.

The microscopic description showed that of the six sections, four were composed of hyperplastic cartilage, fibrocartilage, and scattered newly formed bone trabeculae and periosseous tissue. There was a marked overproduction of immature cartilage and pre-cartilagenous and perosseous tissue. All layers except the bony trabeculae showed more than normal activity. One section was made up of striated muscle and surrounding fibroadipose tissue. It showed edema and scattered leucocytic infiltration with a few polymorphonuclears in some areas. A section of skin and subcutaneous tissue showed a marked fibroblastic activity beneath a thin squamous epithelial layer. The layer was infiltrated with leucocytes and endothelial leucocytes with a very dark-staining cytoplasm. Often these cells contained multiple nuclei up to large foreign body giant cells. The deep edge

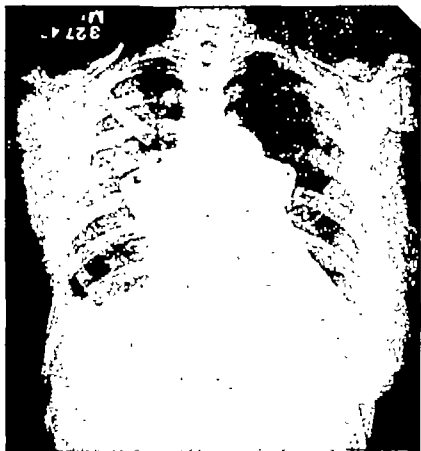


Fig. 3 (Case 1).—A postoperative roentgenogram showing the tantalum plate in position.

of the section showed the wall of an abscess cavity containing numerous macrophages and foreign body giant cells. The diagnosis was osteogenic sarcoma of sternum (osteochondrosarcoma).

The patient's postoperative course was uneventful. There were no pulmonary complications, and she was out of bed on the third day.

Oxygen was given for the first twenty-four hours as a routine measure. There was considerable serous drainage during the first week which subsequently became much less but persisted in small amounts. The patient was kept in the hospital until April 14, 1947, so that she could be carefully observed, after which she was followed in the Tumor Clinic. Her general condition showed very definite and progressive improvement, but as had been anticipated, a moderate amount of serous drainage persisted.

She was readmitted to the hospital on June 24, 1947, for the purpose of removing the tantalum plate. It was decided that enough time had elapsed for a satisfactory inflammatory membrane to have developed. The operation merely consisted of elevating one side of the flap, cutting the wire sutures, and sliding out the plate. With the flap lifted up, it was interesting to note that the foreign body reaction had created a firm, unyielding inflammatory membrane which allowed no motion with respiration. The mam-

The operation consisted of wide block excision of the tumor. In order to accomplish this it was necessary to resect portions of the third, second, and first ribs on both sides, as well as the inner aspect of both clavicles, and to transect the sternum at the level of the third interspace. In the neck, the muscle attachments to the clavicle were divided through a transverse incision across the suprasternal area. The tumor was found to bulge into the anterior mediastinum, where it was adherent to the innominate veins, from which it was separated by sharp and blunt dissection. It was then possible to remove this entire segment of the anterior chest wall with the tumor. During the dissection, the pleura on either side was reflected so that neither pleural cavity was entered. A tantalum plate, which had previously been perforated to allow for the escape of serum, was cut to fit the major portion of the defect and sutured with No. 38 wire to the cut ends of the second and third ribs on either side and to the sternum.



Fig. 2 (Case 1).—Roentgenogram of tumor. An oblique view showing destruction of manubrium and invasion of the sternoclavicular joints.

A large pedicle flap was then raised which included the entire right breast with the underlying pectoralis major and its fascia. This was rotated upward and sutured into the defect. One small rubber tissue drain was inserted beneath the pedicle in the lower portion of the wound. By undermining, it was possible to close the major portion of the area from which the pedicle had been raised, leaving only a small area to be skin grafted. During the course of the operation 1,000 c.c. of blood was given. The patient's condition was excellent at the completion of the procedure.

The pathologic report was as follows: The specimen consisted of an elliptical piece of skin measuring 14.5 by 7.5 cm., in the center of which was a scar 2 cm. in length. Attached to the skin was a section of sternum 10 cm. in length, right and left clavicles measuring 4.5 and 7 cm., respectively, and both right and left, first, second, and third ribs were attached to the sternum. Skin and subcutaneous tissue were removed in toto. There was a swelling 5 by 4.5 cm. which included the left clavicle and a small portion of the right sternoclavicular junction. It was gray white, firm, and covered by a thin, fibrous capsule. On the undersurface of the sternum there was an additional swelling 4.5 by 2 cm. It was brownish red, soft, and rubbery and covered by a thick, fibrous capsule. Cut sections revealed a spherical mass 4.5 by 4 cm. The surface showed pearly, white, soft, gelatinous areas with streaks of gray-white to white-yellow calcified material. There were

brane had formed on the anterior and posterior surfaces of the plate, but of the two, the mediastinal surface appeared to be the more rigid. The incision was closed with interrupted silk.

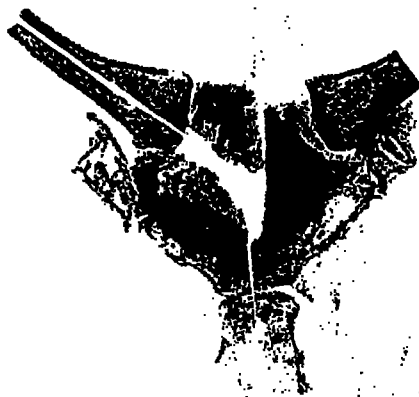


Fig. 6 (Case 1).—Roentgenogram of specimen indicating the extent of the resection.



Fig. 7 (Case 1).—Final result showing defect closed with a pedicle flap which includes the right breast and the underlying pectoralis major muscle with its fascia.

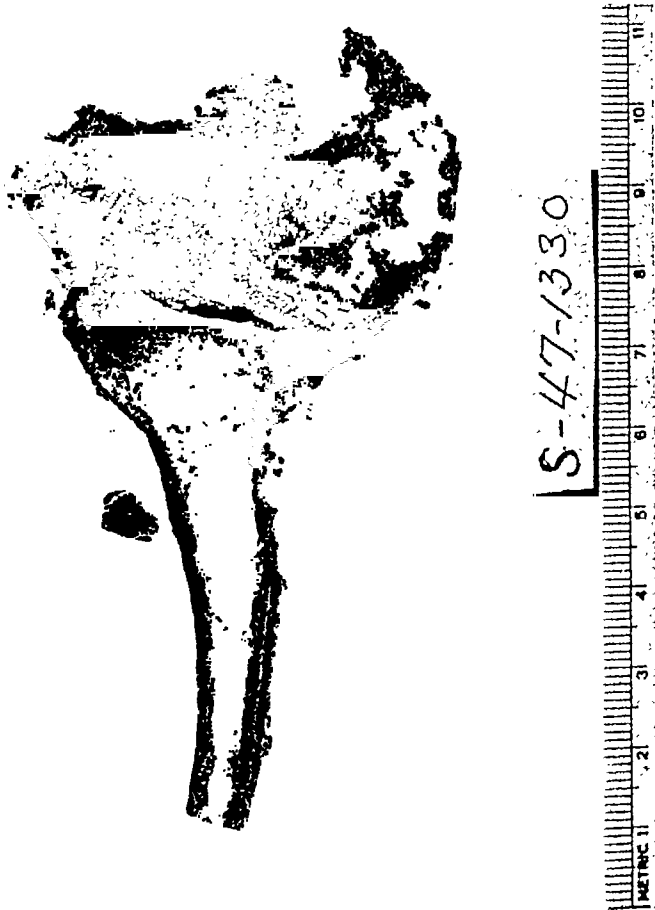


Fig. 4 (Case 1).—Section through tumor and sternum. Note destruction of manubrium.

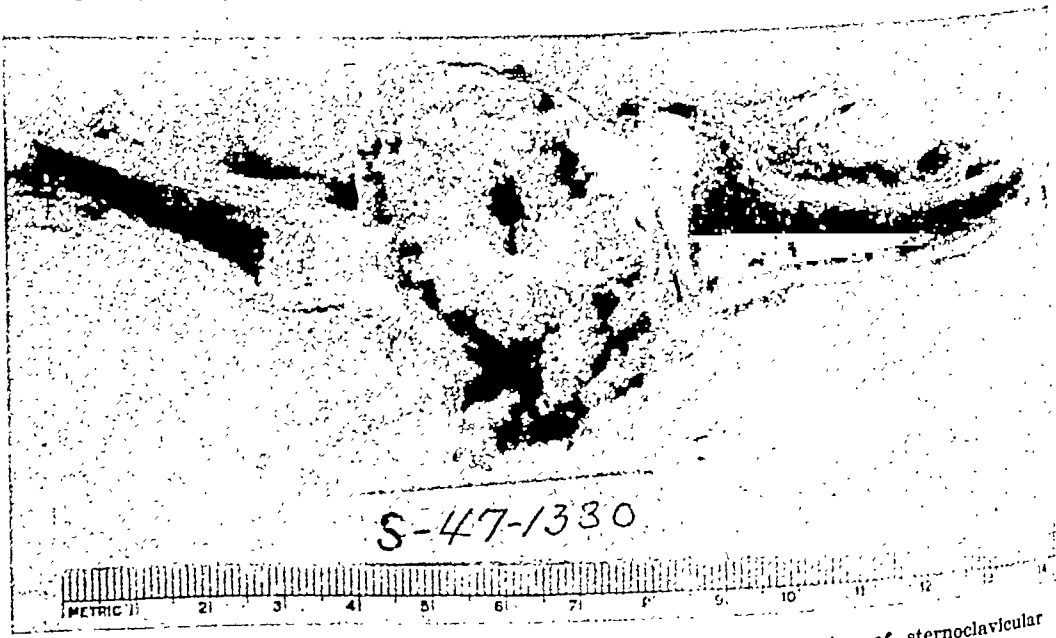


Fig. 5 (Case 1).—Section through tumor and clavicles showing invasion of sternoclavicular joints.

mediastinum. The diaphragms were normal. There was no effusion. The conclusions were that the findings were characteristic of a malignant neoplasm of the left lung with extension to an involvement of the adjacent seventh rib and pleura.

The patient was placed on aerosol penicillin four times a day, and the operation was performed on May 6, 1948.

A right-angled incision was made from the fourth to the eighth ribs paravertebrally and along the eighth interspace to the posterior axillary line. The soft tissue flap was elevated and the ribs exposed at their spinal junctions, after which the fifth, sixth, seventh, and eighth ribs were divided with the intervening, intercostal bundles. A long incision was then made through the fourth interspace, and the pleural cavity opened. It was now possible to make a careful exploration of the lung and the tumor. The latter was found to be adherent to the sixth, seventh, and eighth ribs but had no pleural attachments beyond this area. The incision through the fourth interspace as well beyond the limits of the tumor from above. It appeared to arise from the apex of the lower lobe and was adherent to the lower surface of the upper lobe posteriorly. There was no obvious invasion of glands around the lung root. The mediastinal pleura was incised, and the artery and vein to the lower lobe were dissected out, doubly ligated, and divided, after which the bronchus was isolated and cut across and closed with four interrupted sutures of No. 30 cotton. The mediastinal pleura was then closed with interrupted No. 30 cotton sutures. With the lower lobe divided, it was then possible to rotate it laterally, and a clear exposure of the attachments of the tumor to the chest wall could be obtained. The excision was completed by incising through the eighth interspace and dividing the fifth, sixth, seventh, and eighth ribs in the posterior axillary line and including the lower portion of the upper lobe in the excision. The entire specimen was then removed en bloc. The large defect in the chest wall was closed with a tantalum plate, which was fashioned to fit the defect and was fixed in place with wire sutures after drilling the fourth and ninth ribs at intervals of 2 cm. and the stumps of the cut ribs proximally and distally. One cigarette drain was placed on the surface of the plate and brought out at the lower portion of the wound which was closed in layers. The upper lobe was re-expanded as the chest wall was closed. One catheter was inserted in the dependent part of the thorax.

The pathologic report was as follows: the specimen consisted of a lower lobe with attached portions of four ribs. The diaphragmatic surface was not unusual. The bronchial tree in the lower portion was dilated with thickened mucosa and transverse markings. Binding the four ribs firmly to the surface of the lung was a tumor mass 7 by 4.5 by 5 cm. which had been incised. The surface peripherally was gray, hard, and granular, while centrally the tissue was soft and necrotic. The bones were thinned and apparently involved by tumor. Leading from the amputated margin into the central cut surface of the tumor was a thin-walled small bronchus. The bronchus had lost its continuity and disappeared within this tumor mass. No other bronchi were seen to traverse the tumor tissue. On section the tumor had a gray-tan, granular surface. The pulmonary arteries were not involved in the tumor mass. The portion of tumor invading the bone was firm, rubbery, and elastic. Areas containing greenish-yellow puriform material measuring up to 4 mm. in greatest diameter were seen scattered throughout the tumor mass. The remaining lung tissue had a yellow-tan surface covered by a wrinkled pleura, which showed numerous red, fibrous tags. It appeared collapsed and on section presented a smooth, non-porous, nonrepitant surface. A portion of another lobe which measured 4 by 2 by 1.5 cm. was adherent to the overlying rib and adjacent lobe.

Section of lung showed replacement of lung parenchyma by cords of anaplastic epithelial cells which had squamous characteristics. There was occasional pearl formation. At the periphery of the tumor areas the alveoli contained clumps of similar appearing cells. Tumor was also seen within a periarterial lymphatic. Section of rib showed invasion by and replacement by infiltrating cords of similar appearing cells. The diagnosis was epidermoid carcinoma of the lung (bronchiogenic) with invasion of pleura and ribs.

The patient's course during the operation was excellent. There was no abnormal blood pressure fall and no suggestion of shock; 1,500 c.c. of blood were given. He was allowed up on the second postoperative day.

After removal of the plate there was prompt healing of the wound. The patient has been followed at regular intervals at the Tumor Clinic, the last visit being on Jan. 12, 1949, at which time she was free of symptoms and carrying on her usual activities.

CASE 2.—W. M., a 72-year-old white man, was admitted to the Rhode Island Hospital on April 29, 1948, with a complaint of cough and pain in the left anterior part of the chest of about one month's duration which had been growing steadily worse. He had had no expectoration or hemoptysis, and although he complained of fatigue, he had noticed no loss in weight.



Fig. 8.

Fig. 9.

Fig. 8 (Case 2).—Preoperative roentgenogram of chest.

Fig. 9 (Case 2).—Postoperative roentgenogram with tantalum plate replacing the bony defect.

Examination revealed an elderly man who appeared chronically ill. There was moderate sclerosis of the peripheral arteries, but his heart was essentially normal to physical and electrocardiographic examination. Blood pressure was 156/84. The breath sounds and tactile fremitus were slightly decreased throughout the left side of the chest. There was moderate dullness noted in the mid-chest region anteriorly and posteriorly, and fist percussion on the left caused slight pain. No râles were present. General examination was otherwise not remarkable.

Red blood count was 5.01, hemoglobin 15, and white blood count was 15,150 with 63 per cent polymorphonuclears. Blood urea nitrogen was 17, glucose 81, and protein 6.6. Hinton test was negative. Bronchoscopy on April 20, 1948, was entirely negative. No tumor was seen, and no abnormal secretions were noted.

The report of the x-ray examination of the chest on April 19, 1948, was as follows: the roentgenogram showed a roughly rounded, irregular density in the mid-portion of the left lung field, extending from the seventh to the ninth ribs posteriorly. On the lateral view the mass appeared to be situated well posteriorly, apparently in the apex of the left lower lobe. There was definite erosion and beginning destruction of the inferior border of the posterior axillary portion of the seventh rib. Other portions of the lung were clear. The cardiovascular outlines were normal. There was no shifting of the trachea, heart, or

were required each day. There was no complaint of cough and no history of expectoration or hemoptysis. Roentgenogram of the lung fields was negative, and there was no evidence of destruction of the ribs.

He had had a mild cerebral accident about seven months previously. At that time some weakness of the left part of the face, left arm and leg were noted, but there had been no progression of these signs.

Examination revealed a white man who showed evidence of marked weight loss. There was a tumor palpable between the spine and the medial border of the right scapula about 6 by 5 cm. It was firm and irregular in outline and appeared to be firmly attached to the chest wall at about the level of the fifth and sixth ribs. It was slightly tender to palpation. Examination of the lungs revealed normal breath sounds and no dullness, and only slight limitation of expansion on the right. The heart sounds were normal, and the blood pressure was 110/80. There was slight facial asymmetry due to a left facial weakness. Knee jerks on the left were diminished. Babinski sign was negative, and no sensory changes were present. A slight weakness of the left leg was noted but no paralysis. Apart from general evidence of weight loss, physical examination was otherwise normal.

Laboratory work on entry showed a red blood count of 3,76, a normal urine, and a hemoglobin of 12.1 Gm. The blood urea nitrogen was 24, glucose 77, and the blood protein 4.7 mg. per 100 c.c.

X-ray examination of the chest revealed no infiltration of the lung fields and no destruction of ribs.

Because of the great severity of the patient's pain it was decided to resect the tumor which appeared to arise from the chest wall, although a primary source in the lung with extension through the intercostal spaces was considered.

Operation was carried out on May 19, 1948. A vertical paravertebral incision was made from the third to the eighth rib, well beyond the periphery of the palpable tumor, and deepened to expose the transverse processes, the third, fourth, fifth, and sixth of which were divided, as were their corresponding ribs and intercostal bundles. The incision was then extended laterally over the seventh rib, which was subperiosteally resected, and the pleural cavity entered. The lung was adherent to the parietal pleura but easily freed up to the region of the tumor where it was firmly adherent to the chest wall in an area about 5 cm. in diameter. The tumor and adjacent chest wall were then free at the posterior and inferior portions of the dissection. The outlined triangular area of skin and subcutaneous tissues was then elevated along with the scapula to expose the ribs in the upper part of the hemithorax.

The pleura was then opened along the line of the second interspace after which the third, fourth, fifth, and sixth ribs were divided in the posterior axillary line with the intervening intercostal bundles and pleura. The severed area of chest wall and the tumor were then completely free with the exception of their attachment to the lung. This appeared to be very superficial, and it could not be definitely demonstrated that there was invasion of lung tissue. However, it seemed wisest to excise a liberal amount of lung tissue rather than to run the risk of entering the tumor. A wedge of lung tissue was therefore included with the specimen which was then removed, and the defect closed with interrupted silk sutures. There was no suggestion of lung invasion at any other point. A palpable gland in the mediastinum was removed and found to be benign.

A tantalum plate which had previously been perforated at several points was cut to fit the bony defect and sutured into place with No. 28 wire. This was accomplished by drilling holes in the periphery of the plate and in the adjacent ribs. The plate was bent to conform to the natural convexity of the thoracic cage in this region. The soft tissues of the chest wall were then closed over the plate with one cigarette drain to the dependent portion of the wound.

The pathologic report was as follows: The specimen consisted of portions of four ribs and apparently included the transverse processes of four vertebrae with attached fibroadipose and muscle tissue. Involving three of the ribs near the vertebral end, there was a firm,

His course was complicated by atelectasis of the left upper lobe with associated cough and temperature elevation. Bronchoscopy was carried out, and a considerable amount of thick mucopurulent secretion was aspirated from the trachea and the left upper lobe bronchus, after which the lobe became well aerated and the symptoms relieved.

There was considerable serous drainage from about the plate which later became seropurulent. This was associated with temperature rise, and it became evident that drainage was inadequate. He was taken to the operating room on June 15, 1948; one side of the flap was elevated and the plate removed. Examination revealed a moderate amount of organized exudate within the space and a firm inflammatory wall on both its surfaces. The wound was closed with one cigarette drain to its dependent portion.



Fig. 10 (Case 2).—Roentgenogram taken Sept. 30, 1948. Note the marked thickening in the area previously occupied by the plate.

Definite improvement continued from this point on. This was rather slow at first, but the temperature subsided, and the drainage at the time of discharge on July 17, 1948, was only slight and was nonpurulent. There has been complete absence of chest wall pain, appetite is good, and he has continued to gain weight. Careful follow-up study was made, and he was last seen on Jan. 3, 1949. He had gained twenty-eight pounds since the hospital discharge and was entirely symptom free. The chest wall was firm and entirely without motion on respiration.

CASE 3.—J. H., a 58-year-old white man, was admitted to the Rhode Island Hospital on May 13, 1948, with a chief complaint of severe pain in the right side of the chest. He began to have pain in the right anterior part of the chest and right axilla six months prior to admission. It began rather suddenly in November, 1947, and grew gradually worse, and became constant. He was admitted to another hospital for observation in April, 1948. He had lost considerable weight and the pain had become so severe that large amounts of sedation

satisfactory appearance of the chest wall wound, the patient showed no evidence of weight gain. There appeared to be a gradual decline in his general condition in spite of a fair appetite, absence of pulmonary symptoms, and ability to be out of bed daily. He was discharged from the hospital on July 16, 1948, and follow-up study was made at home by his family physician. He died on Sept. 12, 1948.

COMMENT

The preceding cases, although presenting individual problems, all required extensive removal of the supporting framework of the thoracic cage. Unless favored with mediastinal fixation this may result in difficulties which can be serious in the immediate postoperative period. Attempts to neutralize the adverse effects of a swinging mediastinum, such as may be observed following extrapleural thoracoplasty, by means of external appliances or by packing and strapping of the wound have not always been successful. In cases where it is necessary to remove the periosteum with the ribs no stiffening of the chest wall can be hoped for, and although recovery from operation may occur, a flabby and mobile chest wall invariably follows. The use of tantalum plate in the manner described has obviated these difficulties.

Multiple perforations were made in the plate in one patient (Case 1), who was the first to be operated upon. This was so successful that the same type of plate was used in Cases 2 and 3, where a somewhat different problem was presented. In the latter instances the lung expanded against the plate, and in both cases an abnormal amount of reaction occurred. The reason for perforating the plate in Case 1 was to allow for drainage of serum from the anterior mediastinum. Adequate drainage from the pleural cavity can, of course, be effected by means of a catheter in a dependent position.

In the future a smooth plate will be used when dealing with lesions involving the lung. This is particularly applicable when only a lobectomy has been done and the remaining lobe must expand against the plate.

When in addition to removal of the bony structures a wide resection of the skin and other soft tissues must be carried out, large lateral pedicle flaps are most useful. On the anterior surface in the female, the breast is ideal for this purpose, but regardless of the location of the donor site the pedicle should include muscle and fascia in order to give proper stability. By undermining, the major part of this defect can be closed and the remainder covered with a split-skin graft. The inflammatory wall created by the foreign body reaction completes the formation of a rigid chest wall. The final result bears a close resemblance to that obtained following a Schede type of thoracoplasty for chronic empyema.

It is believed that the dramatic relief of pain in Case 3 which allowed this patient to live out his remaining days in comparative comfort, justified the operation.

SUMMARY

Three cases are presented in which tantalum plate has been used to avoid complications which occur when a large section of the chest wall has been removed. This has had the effect not only of overcoming postoperative difficulties but, in addition, has given stability to the area of excision.

pinkish-tan, seemingly localized tumor measuring 1.5 cm. in greatest diameter which blended at its margins with the surrounding tissue. On section the tumor retained a sharp cut edge. It had a homogeneous mucosa-appearing surface.

Attached to the inner rib aspects there was a greenish-gray portion of lung measuring 5 cm. in its greatest diameter. The sections contained carcinoma that apparently originated in the lung and extended to involve the pleura and chest wall. The histology varied. In one section of lung tissue a small bronchus was involved. In some sections the cells lined alveolar spaces, giving an adenoid appearance. These cells were largely squamous in type, but in a few places became tall and columnar, and secreted mucus. There were also numerous small solid groups of squamous cells. Because of the predominance of the squamous histology it was decided to place the neoplasm in that category. The diagnosis was epidermoid carcinoma (squamous cell) of the lung, with extension to pleura and chest wall.



Fig. 11.

Fig. 11 (Case 3).—Showing tantalum plate in position. Its size indicates exactly the extent of the resection.



Fig. 12.

Fig. 12 (Case 3).—Postoperative roentgenogram taken July 9, 1948.

The patient made an uneventful recovery from the operation. There was never any evidence of shock, and in spite of the wide resection of the chest wall there was complete stabilization of the right hemithorax. The patient was allowed out of bed on the third postoperative day. The most striking feature of the postoperative phase was the complete absence of pain. In spite of the fact that practically all sedation was discontinued, the patient was completely comfortable. There was a moderate amount of serous drainage from the wound which later became seropurulent. This was associated with some elevation of temperature, and on June 17, 1948, he was taken to the operating room, the skin flap elevated, and the tantalum plate removed. There was a moderate amount of localized infection on both sides of the plate which appeared to be due to superficial erosion from the lung surface. There was marked rigidity of the tissues which had been adjacent to the plate. After removal of the plate, one cigarette drain was inserted at the dependent portion of the wound. The fever subsided, and during the remainder of his hospital stay there was only slight serous drainage. In spite of the complete absence of pain and the

or five ribs is required, and frequently substantial portions of the sternum must be sacrificed as well. Such a defect frequently extends from the sternum to a point near the anterior axillary line (Fig. 2).

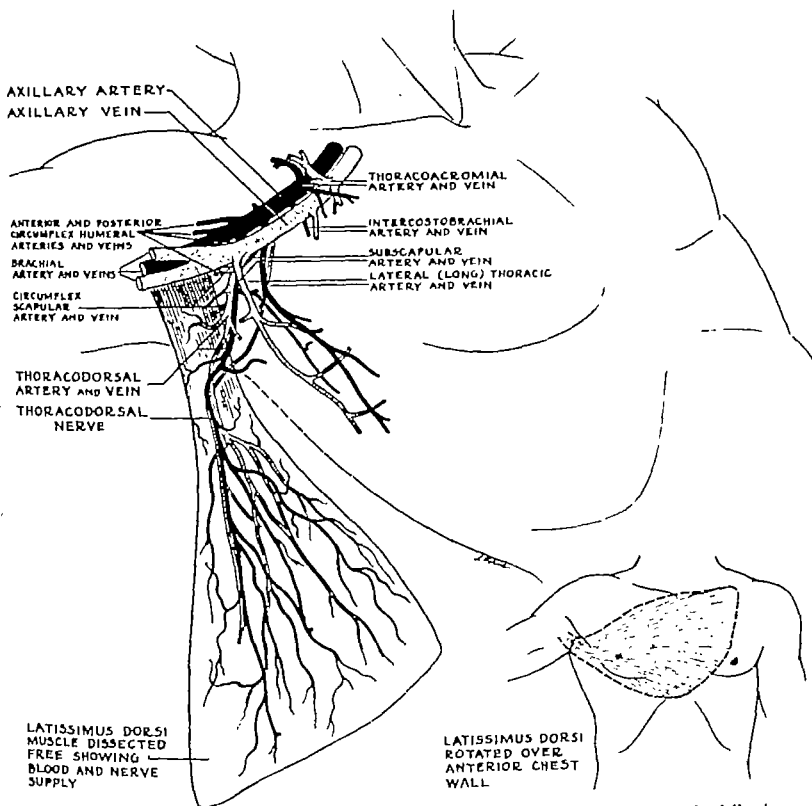


Fig. 1.—Drawing of the latissimus dorsi showing the intact blood and nerve supply following its mobilization.

Fascia Lata Graft.—Watson and James have pointed out the advantages of using a large square of fascia lata in the repair of a large thoracic wall defect (Fig. 3). It furnishes good support to the chest wall and aids in preventing pulmonary herniation. It does, however, require the use of a large pedicled flap of skin and subcutaneous tissue. For large defects this may be hazardous or impossible.

Use of the Latissimus Dorsi.—Various extracostal muscles have long been used in the repair of defects in the lateral and posterior aspects of the chest wall, but no effort has been made to extend their usefulness to include the ante-

RECONSTRUCTION OF THE ANTERIOR THORACIC WALL

DARRELL A. CAMPBELL, M.D., F.A.C.S.

ANN ARBOR, MICH.

THE sacrifice of large portions of the anterior thoracic wall incident to the removal of malignant neoplasms is required in selected cases. These cases include (a) primary neoplasms arising in the bony thoracic wall, (b) recurrent mammary cancer invading the bony thorax, and (c) primary carcinoma of the breast infiltrating the thoracic wall at or near the costal cartilages or lateral aspect of the sternum, where protection from the thoracic wall is not afforded by the interposition of the pectoral muscles.

Although the past decade of progress in surgical technique, physiology, and anesthesia has permitted radical resections of the thoracic wall as well as viscera, few reports have dealt with methods by which these extensive wounds may be repaired. It is the purpose of this report to describe a method which has been satisfactorily utilized in the reconstruction of the anterior thoracic wall.

Relatively small defects in the anterior chest wall can be closed in a number of ways, and among the most satisfactory is the method described by Maurer and Blades¹ which furnishes bony stabilization to the defect. Watson and James² have described the use of fascia lata grafts in the repair of large defects, but these must be covered by large pedicled flaps of skin, for split-thickness grafts can scarcely be expected to survive on fascial patches which are, themselves, free grafts without blood supply. Maier³ has discussed the use of the opposite breast as an aid in the closure of large defects. Pickrell, Kelley, and Marzoni,⁴ in an excellent review of this problem, used pedicled flaps of various design, and in one case in which such a flap was not feasible they applied a split graft directly upon the pericardium. The following procedure which utilizes a fascia lata graft, a completely mobilized latissimus dorsi muscle, and split-thickness skin grafts appears to have advantages not possessed by the foregoing methods.

Excision of the Lesion.—Standards governing the removal of any neoplastic process apply to the excision of these lesions. They include removal of all the neoplasm en bloc, together with an adequate margin of uninvolved tissues. The lymph nodes draining the area occupied by the neoplasm must be removed, if possible. If the operation is being done for recurrent carcinoma of the breast, all the axillary lymph structures will have previously been removed, and this area need not be invaded the second time. Recurrence in the axilla or supra-clavicular area generally constitutes inoperability, and palliation by roentgen therapy is preferable. If the lesion is located over the lateral margin of the sternum, the internal mammary vessels with accompanying lymph structures should be removed.

Resection of lesions infiltrating the bony thorax requires removal of the full thickness of the thoracic wall and rarely will a portion of less than two ribs suffice. In general, a block of chest wall requiring the segments of three, four,

From the Departments of Surgery, Wayne County General Hospital, and University of Michigan Medical School.

Received for publication March 17, 1949.

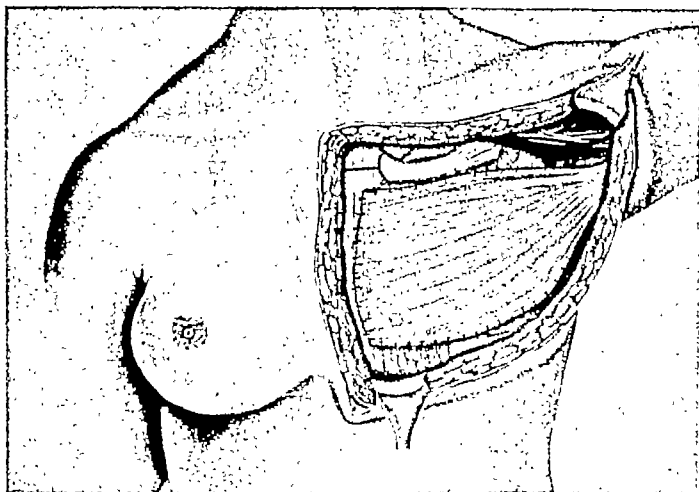


Fig. 4.—Transplantation of the latissimus dorsi to cover the defect.

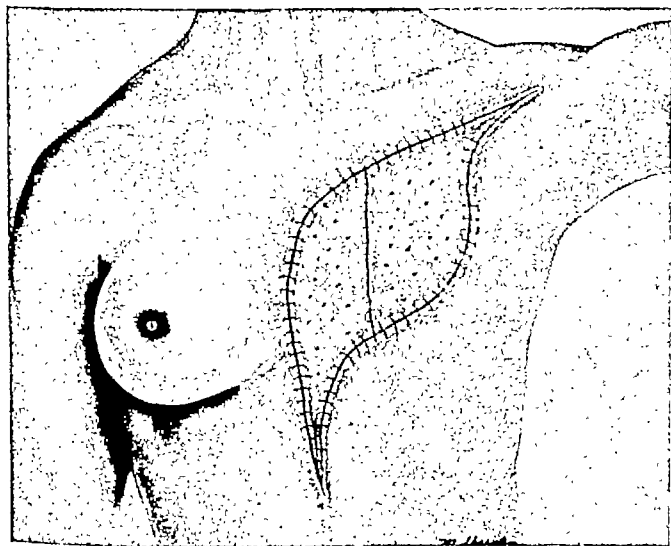


Fig. 5.—Completed procedure following application of split-thickness skin grafts.

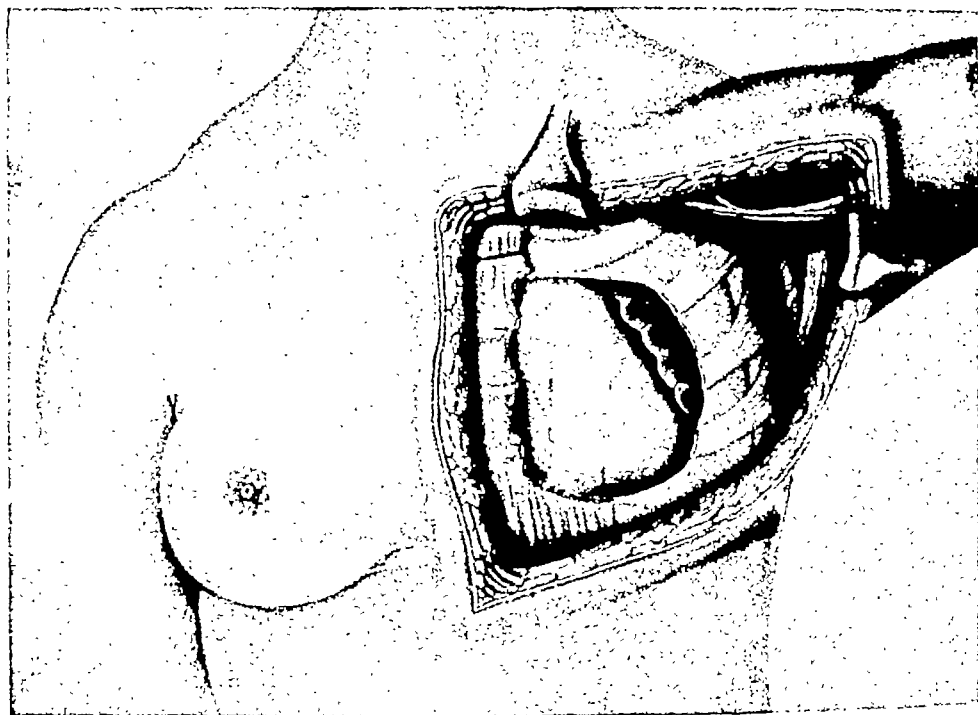


Fig. 2.—Defect of average size following removal of a three-rib segment of anterior chest wall.

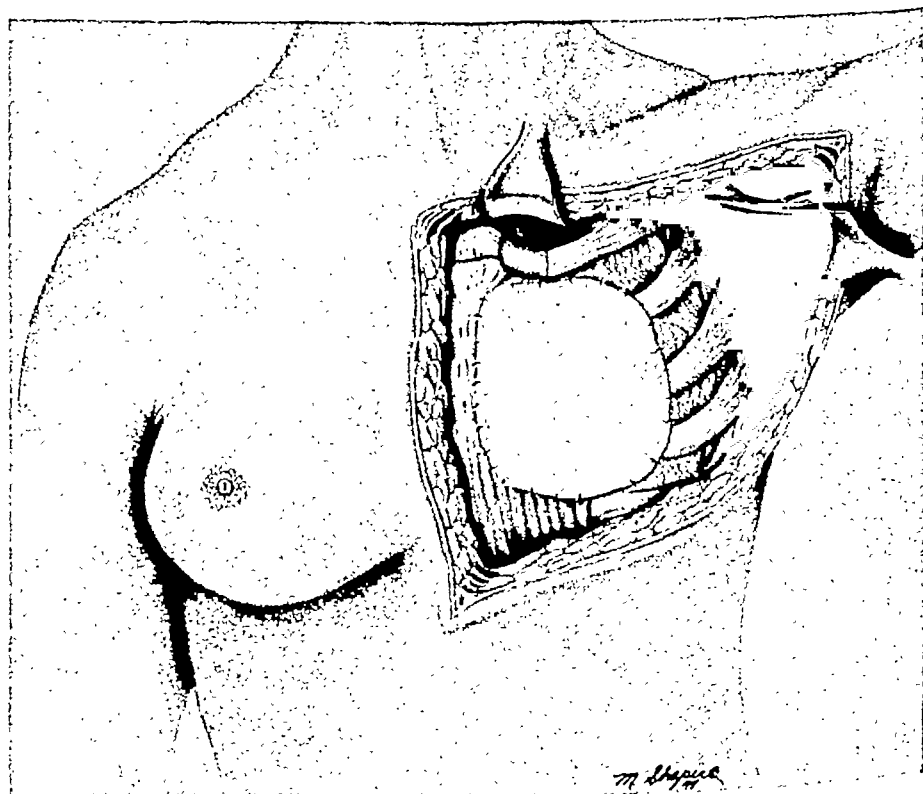


Fig. 3.—Application of the fascial lata graft.

operation. Advantages of the use of this muscle are as follows: (a) It is readily accessible; (b) it can be completely mobilized, except for its insertion, and by means of sharp dissection, ligation of few perforating vessels, and incision of the lumbodorsal fascia which releases it from its origin, small blood loss is encountered; (c) it is capable of reaching any defect on the anterior aspect of the hemithorax; (d) it carries its own vascular and nerve supply during transplantation (Fig. 1); (e) it thus assures adequate nutrition, not only to itself, but to adjacent structures, such as the fascia lata graft beneath and split thickness skin grafts above, which require such assistance for survival; (f) there is little or no functional loss; (g) by furnishing another layer at the site of the defect, it aids in the support of the chest wall.

Some disadvantages may be associated with this procedure. It increases the magnitude of the operation by requiring additional dissection. This appears to be a minor consideration if the conditions for the operation are good. If the operation is being done for recurrent mammary cancer, the surgeon who previously performed the radical mastectomy may have found it necessary to ligate and excise the subscapular vessels. If such is the case, the vascular supply would be almost entirely from the perforating branches of the intercostals, and mobilization of the muscle might leave its viability in question.

Split-Thickness Skin Grafts.—The application of split-thickness skin grafts needs little explanation (Fig. 5). The use of this method to produce an epithelial-covered area seems infinitely safer and easier than that of fashioning complicated pedicled flaps with the associated hazards so well known to most surgeons.

Customary pressure dressings are applied to the skin grafts, and serve also for temporary support to the chest wall for the first few postoperative days. Following their removal, no significant interference with cardiorespiratory function has been noted.

This procedure with various minor modifications has been carried out seven times with one postoperative death. This patient died suddenly sitting at her bedside on the third postoperative day. No autopsy was allowed.

SUMMARY

1. A method of repairing large defects in the anterior thoracic wall has been described. This method utilizes (a) a fascia lata graft, (b) complete mobilization and transplantation of the latissimus dorsi, and (c) split-thickness skin grafts.

2. Some advantages and disadvantages of this method are discussed.

REFERENCES

1. Maurer, E., and Blades, B.: Hernia of the Lung, *J. THORACIC SURG.* 15: 77, 1946.
2. Watson, W., and James, A.: Fascia Lata Grafts for Chest Wall Defects, *J. THORACIC SURG.* 16: 399, 1947.
3. Maier, H.: Surgical Management of Large Defects of the Thoracic Wall, *Surgery* 22: 160, 1947.
4. Pickrell, K. L., Kelley, J. W., and Marzoni, F. A.: The Surgical Treatment of Recurrent Carcinoma of the Breast and Chest Wall, *Plast. & Reconstruct. Surg.* 3: 150, 1948.

rior aspect of the thorax. The latissimus dorsi, however, is capable of being transplanted to the anterior chest wall to assist in the repair of a defect at this site (Fig. 4). Exposure of the muscle is not difficult. If the excision of the neoplasm requires the dissection to extend to the anterior margin of the muscle, it can be mobilized by sharp dissection without additional skin incision. It is of some assistance if the patient is placed slightly on the opposite side prior to

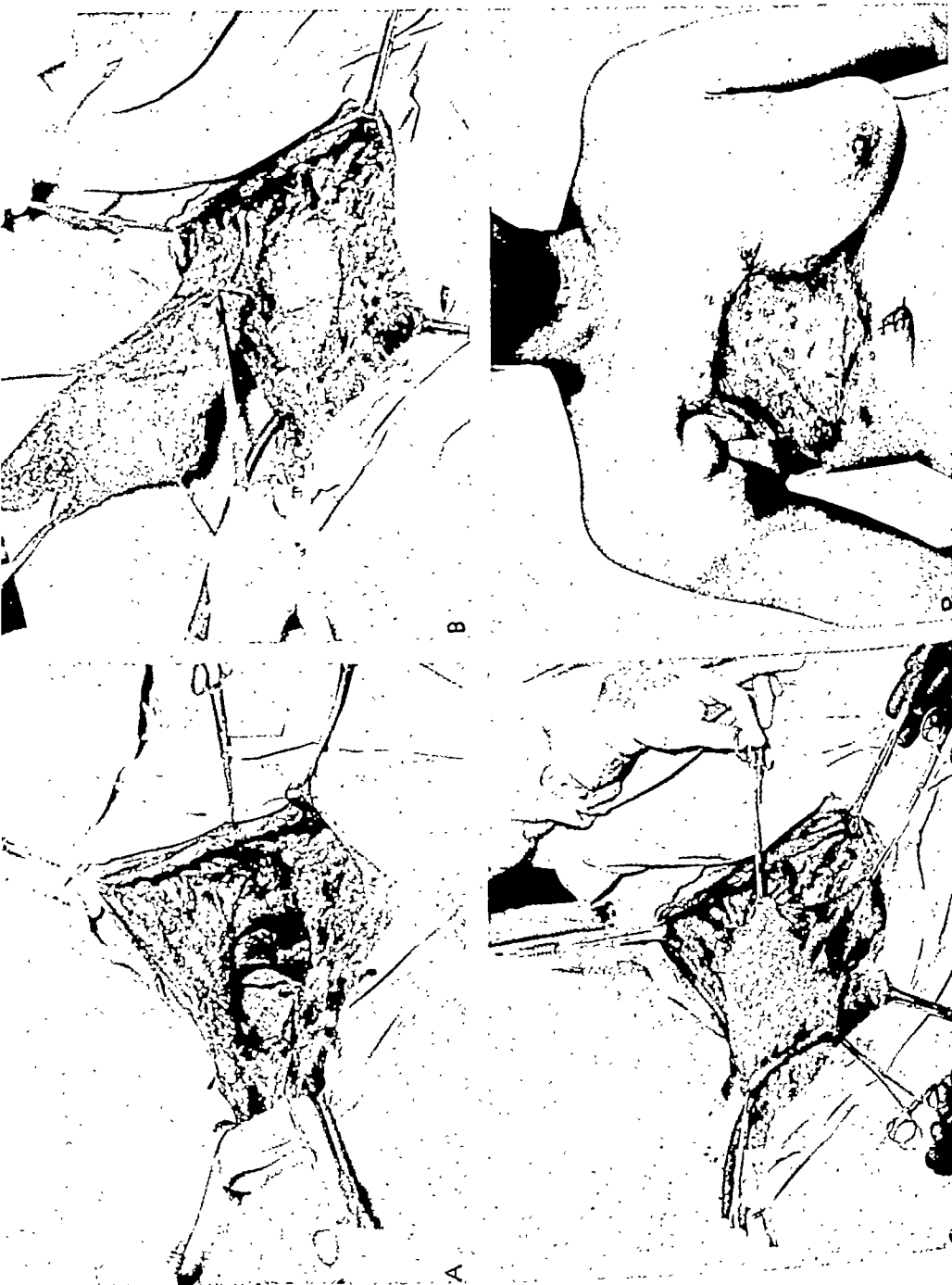


FIG. 6.—Photographs showing A, excision of lesion; B, tumor into graft in place and latissimus dorsi mobilized (blood supply to muscle demonstrated by pointer); C, transplantation of muscle; D, tumor into graft in place and latissimus dorsi mobilized (blood supply to muscle demonstrated by pointer).

dition, the submucosa in dogs contains more mucous glands and fewer elastic fibers. These differences are minor, however, and the esophageal tissue of both man and dog is sufficiently similar in texture and consistency for surgical comparison.

For the sake of clarity, definitions of some of the terms used to describe the function of the esophageal muscle are given. Tonus is the resistance which the smooth muscle of the esophagus offers to distention.¹² Primary peristalsis is a progressive wave of contraction, carrying a bolus from the pharynx to the stomach. Secondary peristalsis, or segmentation, is a rhythmic nonprogressing contraction, not propelling food, and unrelated to tonus.

EXPERIMENTS

Operations were performed on sixty-three adult mongrel dogs of both sexes, ranging in weight from 12 to 30 pounds. In all experiments sodium pentobarbital was used for anesthesia, and pulmonary ventilation was maintained with intermittent positive pressure air insufflation given through an endotracheal tube. A right cervical and several different transpleural approaches were employed, depending on how large a portion of the esophagus was to be separated from its bed. In all instances the main trunks of the vagus nerves were visualized and dissected free from the esophagus. The actual mobilization of the esophagus was accomplished by sharp and blunt dissection. All possible delicacy was employed in handling the esophagus with instruments. Blood vessels, except for the azygos vein, were ligated only if they bled copiously.

The experimental series was divided into six groups, shown diagrammatically in Fig. 1.

Group I had an extensive procedure through a combined thoracocervical approach which permitted division of the esophageal blood and nerve supply between the cricoid cartilage and the diaphragm. *Group II* underwent a similar operation, except that the 2.0 cm. portion distal to the cricoid cartilage and another 2.0 cm. portion proximal to the diaphragm were left undisturbed. In *Group III* devascularization and denervation were limited to the thoracic esophagus from the diaphragm to a point about 2.0 cm. above the aortic arch. *Group IV* had the thoracic esophagus mobilized from the aortic arch, through the diaphragmatic hiatus, to the cardia. In this procedure the vessels extending 3.0 cm. along the greater and lesser curvatures were also divided and ligated. Both vagi were severed at the level of the diaphragm. In *Group V* the esophagus was mobilized between its mid-cervical and mid-thoracic points. In *Group VI* the upper 8.0 cm. of the cervical esophagus was freed from its bed.

Postoperative care included a milk diet for ten days, after which the animals received their usual ground horsemeat and dog biscuit. Follow-up consisted of fluoroscopic and roentgenologic studies two weeks after operation and at bimonthly intervals thereafter, until functional recovery occurred or death terminated the experiment. A thick mixture of barium sulfate, ground horsemeat, and milk, was used as a contrast medium.

Experimental results were evaluated clinically in terms of regurgitation of solids and liquids, and of survival. Objective studies consisted of fluoroscopy, roentgen films, gross and microscopic examination of specimens stained post mortem by the Loyez myelin, eosin-thionin, and eosin-cresyl violet techniques. The specimens were also studied by arterial injection with latex,* using a modification of Demel's method.¹¹ These were cleared in methyl salicylate and photographed by direct and reflected light.¹³

Our method of injecting the arterial system was as follows.

1. Anesthetize and exsanguinate dog.
2. Cannulate the abdominal aorta with a No. 13 needle below the renal arteries and secure it with three heavy ligatures.
3. Incise both external jugular veins and the inferior vena cava.

*Latex; 8-765-A Red No. 1.

BLOOD AND NERVE SUPPLY TO THE ESOPHAGUS

AN EXPERIMENTAL STUDY

ORVAR SWENSON, M.D., KEITH MERRILL, JR., M.D., E. CONVERSE PEIRCE II, M.D.,
AND HAROLD F. RHEINLANDER, M.D.

BOSTON, MASS.

THE experimental study of the blood supply to the dog's esophagus was prompted by statements in the literature on esophageal surgery that the circulation of the esophagus is poor.¹⁻⁵ A recent study of end-to-end anastomosis of the esophagus under tension⁶ demonstrated that considerable portions of the thoracic esophagus could be resected with extensive freeing of each segment, and successful end-to-end anastomosis performed. The present experiments attempted to define safe limits of mobilization of the esophagus in an effort to extend the scope of esophageal surgery, particularly in children. The character of the experiments also made it possible to study the effects of esophageal denervation.

The experiments consisted of mobilizing varying portions of the esophagus and evaluating the effect of this on (1) circulation, and (2) function. No comparable study has ever been made, to our knowledge. The nearest approach is the work of de Francesco,⁷ who concluded that surgical denervation of the abdominal portion of the esophagus produced no significant alterations in tone or peristalsis. Although surgical opinion generally considers extensive mobilization of the esophagus dangerous,¹⁻⁵ Miller and Andrus⁸ and Ochsner and Owens⁹ mentioned freeing the thoracic esophagus well up into the mediastinum without serious consequences. Fischer¹⁰ quoted Zaaier as saying that it is possible to isolate the esophagus to a great extent without grave disturbance of its circulation.

The arteries supplying the esophagus of the dog have been described in detail by Saint⁵ and Demel.¹¹ The upper cervical portion is nourished by branches of the thyroid artery. The lower cervical and upper thoracic divisions are supplied by branches from the subclavian artery. The mid-thoracic part of the esophagus receives its blood supply from the bronchoesophageal system, which arises as small branches from the aorta at the junction of its arch and descending portion. The lower thoracic esophagus is nourished by ascending and descending branches stemming from the aorta between the third and fifth intercostal arteries, while the remainder is supplied by diaphragmatic vessels and branches of the left gastric artery. The intrinsic esophageal arteries run longitudinally, and through these the extrinsic vessels of each group intercommunicate freely.

The motor innervation has two components, the pharyngeal branches of the glossopharyngeal nerve and the esophageal branches of the vagus nerve.¹²

The structure of the esophagus in the dog and the human being is roughly comparable, although in the dog striated muscle is more plentiful, comprising the upper third of the circular coat and the entire longitudinal layer. In ad-

From the Department of Surgery, Harvard Medical School, and Surgical Service, The Children's Hospital.

This investigation was supported by funds from the Grant Foundation.

Received for publication March 25, 1949.

4. Flush the circulatory system with two to three gallons of cold isotonic saline solution while manipulating the neck, tongue, and stomach. Continue the flushing until flow from veins is clear and all visible veins contain no blood. The injection pressure should not exceed 250 mm. of mercury.

5. Mix latex* using either PC 520, a white, radiopaque material, or S-765-A red No. 1 for colored preparations; stir carefully in hot tap water and continue dilution until specific gravity does not exceed 1.005.

6. Inject under maximal pressure of 250 mm. of mercury for twenty to forty minutes.

7. Refrigerate entire cadaver for at least twelve hours.

8. Remove injected esophagus, fix in 10 per cent formalin, and clear through alcohol and benzene to methyl salicylate.[†]

EXPERIMENTAL RESULTS

The anatomic results of the esophageal mobilizations are given in Fig. 2. Complete observations on disturbed functions or pathologic findings are summarized as follows.

Group I.—In these eight dogs the mobilization cut off all the extrinsic esophageal blood supply except for the gastric and diaphragmatic branches and some pharyngeal connections (Fig. 1). Denervation included the pharyngeal branches of the glossopharyngeal nerve and the esophageal branches of the vagus.

Three dogs were sacrificed on the third postoperative day to obtain injected specimens. The remaining five died within eighteen days of operation. The average survival time was five days. One subject died of perforation of the esophagus, and the remaining four of pulmonary sepsis secondary to aspiration of saliva and ingested material.

Clinically, all subjects suffered from aspiration of overflowing esophageal contents secondary to cardiospasm and to lack of peristalsis. All began to regurgitate fluids and small samples of food on the first postoperative day and continued to do so until death.

Fluoroscopy of a typical subject two days after operation showed rapid ingestion of contrast medium, normal swallowing movements in the pharynx, but no primary peristaltic wave in the esophagus to grasp the bolus and carry it downward. The barium distended the entire esophagus until the structure filled and overflowed into the larynx. There was intermittent relaxation of the lower 2.0 cm. of the thoracic esophagus, permitting some barium to enter the stomach. In the roentgen films the esophagus resembled an elongated, inert sac with a rather uniform diameter of 3.0 cm. (Fig. 3).

Autopsies in this group revealed one mucosal ulceration, and one esophageal slough which encircled the esophagus and extended 3.0 cm. above and 7.0 cm. below the first rib. Microscopic examination of tissue near the slough showed acute inflammation with necrosis.

Summary.—Two dogs developed mucosal ulcerations and in one there was esophageal perforation. Aspiration and fatal pulmonary sepsis caused the death of four out of five unperfected dogs. In this group, neuromuscular impairment overshadowed vascular damage. Denervation caused cardiospasm as well as dilatation and loss of peristalsis in the esophageal segment mobilized.

Group II.—These twelve dogs had a mobilization of the esophagus which interrupted the blood supply from the subclavian, bronchoesophageal, and the upper aortic branches, and a denervation involving the vagus fibers from 2.0 cm. below the cricoid cartilage to 2.0 cm. above the diaphragm.

Fatal sequelae of devascularization were three perforations and three strictures of the esophagus. One dog died of pneumonia two days after operation. Another dog died of wound sepsis six days after operation. Four dogs lived to recover function. The stricture gradually disappeared in one subject.

*Stock solutions sold by General Latex and Chemical Company, Cambridge, Mass.

[†]Time schedule for clearing: 70 per cent ethyl alcohol twenty-four to thirty-six hours; 95 per cent ethyl alcohol for another twenty-four to thirty-six hours; absolute alcohol, with two changes, twenty-four hours total; benzene U.S.P. twenty-four to forty-eight hours; store in Methyl salicylate, U.S.P., four parts, to benzyl benzoate, U.S.P., three parts, until clear.

REGIONS MOBILIZED
in the
VARIOUS GROUPS

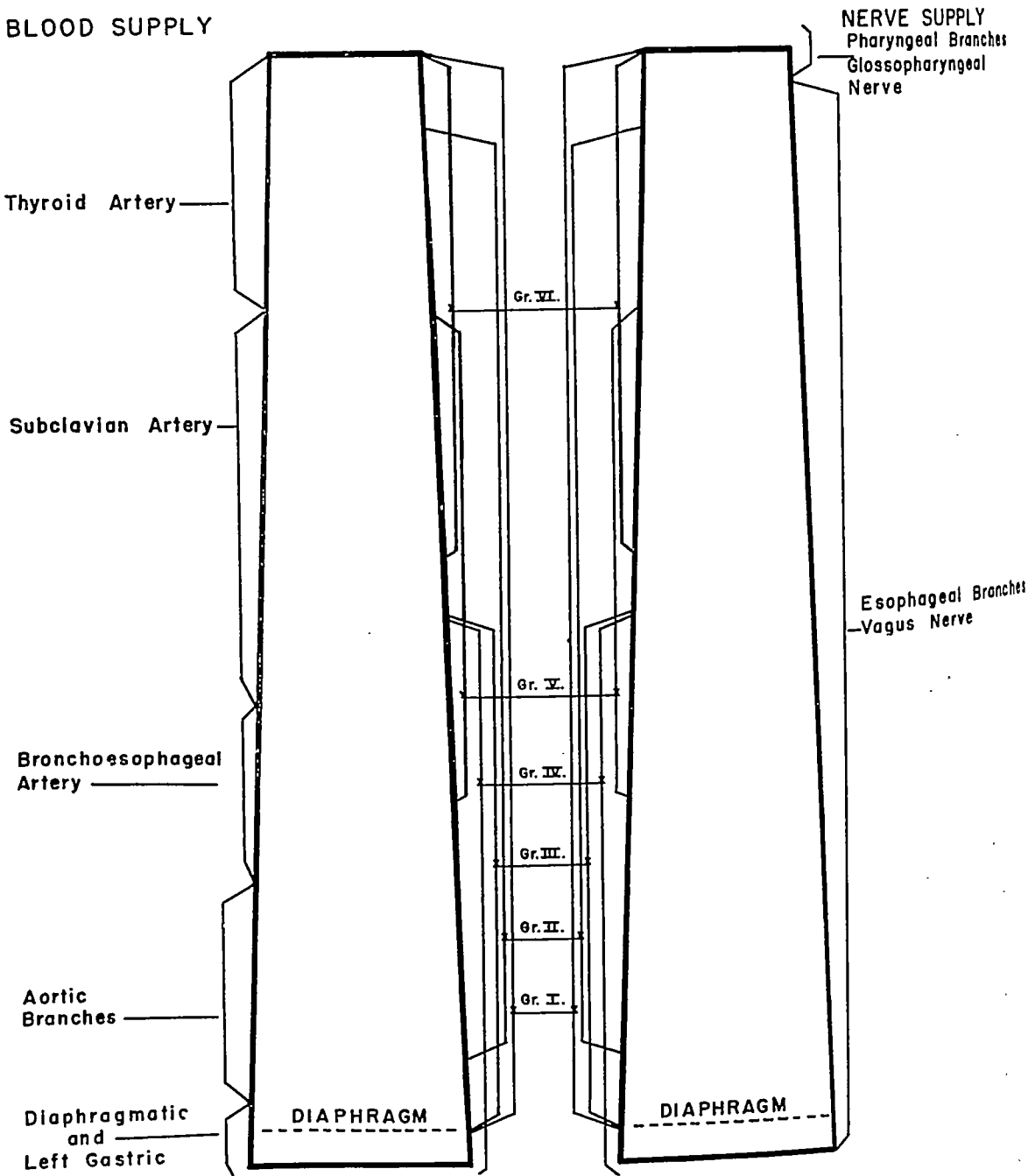


Fig. 1.—Regions mobilized in the various groups.

Regurgitation of solids occurred in eight animals, and in three fluids and saliva also were regurgitated. Three of these subjects developed strictures severe enough to cause death.

During fluoroscopy the contrast medium would be ingested and readily swallowed until the esophagus resembled an elongated, smooth-walled fusiform sac. Both the cervical and thoracic portions were involved, indicating lack of tonus.^{14, 15} If more food were given it would push the esophageal contents through the cardiac sphincter into the stomach. If food were withheld, the dog would either regurgitate the esophageal contents, or else the latter would sluggishly enter the stomach during intermittent relaxation of the cardiac sphincter over a period of five to ten minutes.

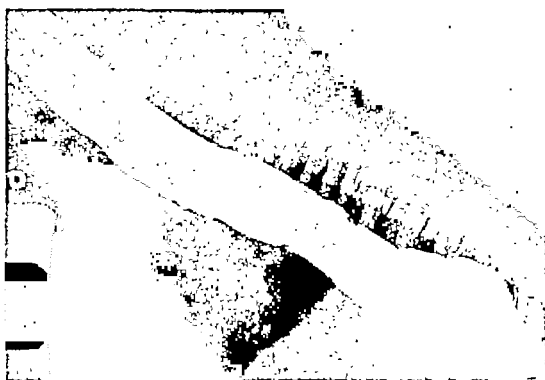


Fig. 3.—Lateral film two days after operation on dog in Group I. Note marked dilatation and absence of peristaltic waves.

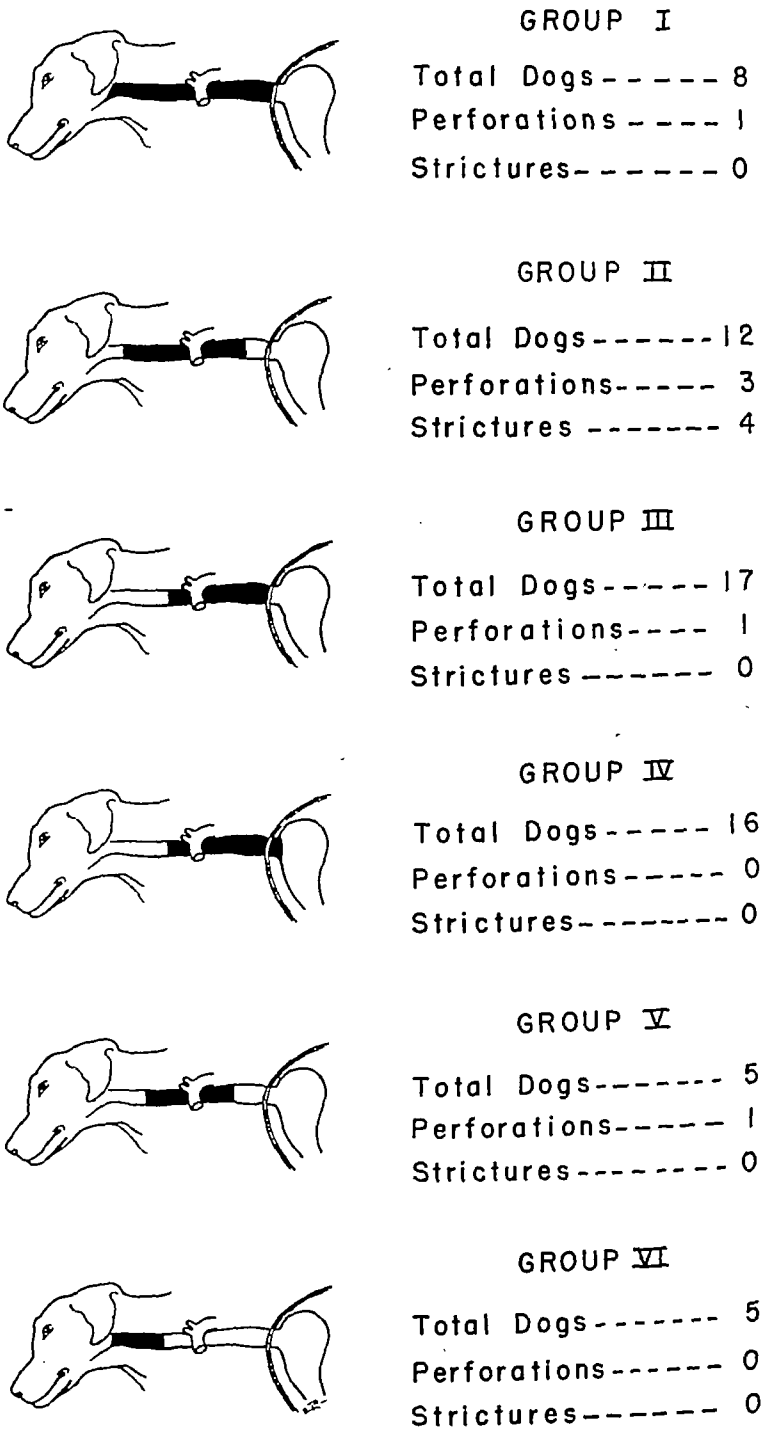
The dilatation was temporary. The diameter of the cervical esophagus, normally 1.5 cm., increased to a maximum of 3.3 cm. at approximately six weeks after operation. During the same time the thoracic portion, normally 2.0 cm., attained a maximal diameter of 3.5 cm. (Fig. 4).

Effective peristalsis reappeared approximately ten weeks after operation, when it swept boli 5.0 to 6.0 cm. long down the esophagus to the stomach. Completely normal primary peristalsis was not present until about twenty-four weeks after operation, when boli were reduced to a normal 2.0 to 3.0 cm. and were carried without delay to the stomach.

Minimal cardiospasm was seen in both films and fluoroscopy and disappeared after peristaltic recovery. It seemed more closely related to the absence of primary peristalsis than to true spasm, although it resembled Templeton's¹⁶ description of cardiospasm.

Roentgenologic evidence of stricture at the level of the first rib appeared in four dogs within two weeks of operation. Each stricture consisted of a narrowed area 1.5 to 3.0 cm. long. One stricture disappeared gradually over a period of six months. In the other three dogs, stricture was severe enough to result in fatal complications.

Two animals, at necropsy six days after operation, were found to have esophageal necrosis with perforation. A third, sacrificed eight weeks after operation, showed a small, walled-off perforation and fibrous replacement of muscular and submucosal layers. Specimens from the three animals with strictures resulting in fatal complications exhibited an even more striking fibroblast proliferation into all layers, with degeneration of the epithelium to a one-layer cuboidal type. The strictures and perforations were found within 1.0 cm. of the first rib.



Devascularized areas shown in solid black

Fig. 2.—Anatomic changes following mobilization of various portions of the esophagus.

Solid food was regurgitated by four dogs and fluids by two, from the seventh to the eighteenth postoperative day, with one exception. This animal continued to regurgitate, with intermittent aspiration of food and saliva; death from pneumonia and multiple lung abscesses finally occurred three months after operation.

At fluoroscopy, the subjects had normal primary peristalsis to the level of the first rib. Below this point there was no peristalsis, and the esophagus dilated to a fusiform shape nearly three times its normal diameter. After the entire esophagus was fully distended, intermittent relaxation of the cardia occurred, sufficient to allow small boli to pass, but complete emptying did not take place. Gradual return of tonus began at about ten weeks, after the thoracic esophagus had reached a maximal diameter of 5.0 cm.

Beginning two weeks after operation, primary peristalsis and spasm of the lower 2.0 to 3.0 cm. of the esophagus were seen on all films. Primary peristalsis was too weak to be demonstrated fluoroscopically until approximately ten weeks after operation. Segmental contractions were minimal in number and strength two weeks after operation. These gradually increased in depth, but were too weak to be visualized by fluoroscopy until four to six weeks after denervation (Fig. 5).



Fig. 5.—Lateral film of thoracic esophagus taken ten weeks after operation on dog in Group III. Note dilatation, absence of primary peristalsis, and cardiospasm.

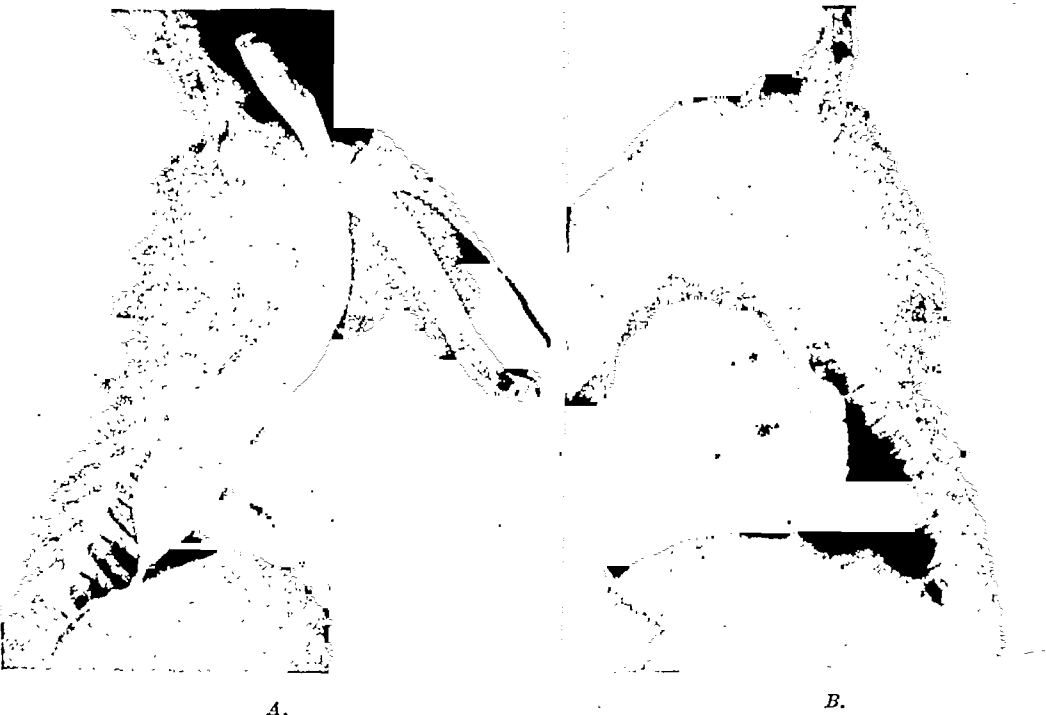
Functional recovery occurred twelve weeks after operation in one dog. Another, who survived a similar length of time, had cardiospasm sufficiently severe to cause chronic aspiration. A third long-time survivor at sixteen weeks showed strong primary peristalsis, insufficient to overcome severe cardiospasm. At twenty weeks diminishing cardiospasm and dilatation were seen. At twenty-four weeks good function, with return of tonus and disappearance of cardiospasm, was demonstrated. At twenty-six weeks primary peristalsis transported boli 10.0 to 12.0 cm. long down the esophagus at normal speed without either residue in the upper thoracic esophagus or delay at the cardia.

At necropsy, severe circulatory insufficiency was manifested in one esophagus which perforated at the level of the first rib five days after operation. Ulcerations of the mucosa, up to 6.0 cm. in length, were observed in five specimens examined within two weeks of operation. Since they were found after the date at which perforation was usually seen, we considered them as phenomena of secondary importance.

Gross evidence of neuropathologic change was a striking flabbiness and dilatation of the thoracic esophagus. The circumference ranged from an average of 3.0 cm. three days after

Four animals showing complete peristaltic recovery were sacrificed twenty-eight, thirty, thirty-four, and thirty-eight weeks after operation. All specimens were grossly normal in color and circumference, and were lined with healthy epithelium. Microscopic examination of sections taken at the levels of the first rib and the cardia showed completely normal structure.

The gross changes resulting from denervation were a marked dilatation and flabbiness of the esophagus. These were present in all dogs dying prior to complete peristaltic recovery. The esophageal circumference ranged from 3.0 to 4.0 cm. in the neck and from 4.0 to 5.0 cm. in the thorax, compared to a normal control specimen of similar length whose cervical and thoracic circumferences were 3.0 and 3.5 cm., respectively. This change was observed three days to eight weeks after operation. Normal-appearing ganglion cells of Auerbach's plexus were found in all specimens from dogs with complete functional recovery.



A.

B.

Fig. 4.—A, Lateral film of dog in Group II five weeks after operation. Note dilatation, loss of peristalsis, and cardiospasm. B, Same dog, eleven weeks after operation. Note return of tonus and primary peristalsis.

Summary.—In these twelve animals there were three perforations and four strictures of the esophagus. One stricture disappeared and the dog completely recovered function. Denervation caused temporary cardiospasm as well as dilatation and loss of peristalsis in the mobilized esophageal segment. In the surviving six animals there was no incoordination between deglutition and peristalsis, and cardiospasm was minimal. Primary peristalsis returned in ten weeks and complete recovery occurred twenty-four weeks after operation.

Group III.—In this group of seventeen dogs, mobilization of the esophagus eliminated branches from the lower half of the subclavian artery, the bronchoesophageal arteries, and the aorta. Denervation included vagus fibers from 2.0 cm. above the aortic arch to the diaphragm (Fig. 1).

In this group, one animal died from improper anesthesia; two died in the first postoperative week, one from perforation of the esophagus and the second from wound infection. Two dogs were sacrificed in the first postoperative week to obtain injected specimens of esophagus. Nine died of pneumonia or distemper within twelve weeks of operation; none of these showed functional recovery. Three animals were followed for a longer period of time, and had complete recovery of esophageal function at twelve, twenty, and twenty-two weeks.

Summary.—There were no strictures or perforations of the esophagus in this group of sixteen dogs. Denervation caused temporary cardiospasm and loss of peristalsis in the mobilized esophageal segment. Six of these dogs were studied to determine the time of functional recovery. This occurred in three dogs at twenty-six, and twenty-nine weeks, respectively. In the two which were followed for thirty-four weeks after denervation there was complete recovery, with no abnormalities demonstrable fluoroscopically. One dog showed no recovery of function and was sacrificed during the twenty-eighth postoperative week.

Group V.—In this group of five dogs, mobilization of the esophagus eliminated the ascending and descending branches of the subclavian artery. Denervation excluded vagus fibers from the mid-cervical to the mid-thoracic region (Fig. 1).

There was one death from esophageal perforation. Another death resulted from distemper eight weeks after operation. The three dogs that survived were studied for functional activity.

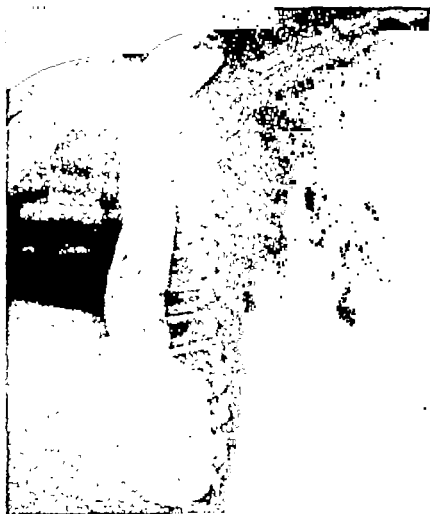


Fig. 6.—Lateral film of thoracic esophagus taken five weeks after operation on dog in Group IV. Note dilatation and absence of primary peristalsis.

None of the subjects showed evidence of food or fluid regurgitation during the immediate postoperative period.

At fluoroscopy the contrast medium was ingested and carried by primary peristalsis to the denervated portion, which dilated to nearly thrice its normal diameter. As long as feeding was continued, ingested boli pushed the contents of the inert, dilated esophagus into the stomach. At the end of feeding the distended area remained free of any peristalsis and emptied gradually over a period of several minutes. Peristalsis was absent approximately six weeks after operation. None of the dogs showed cardiospasm. Functional recovery with normal peristalsis appeared in the three long-term survivors eight to ten weeks after operation.

There was no gross evidence of neuropathologic change.

Summary.—There was only one instance of circulatory insufficiency, manifested by necrosis and perforation of the esophagus at the level of the first rib. Denervation caused dilatation and loss of peristalsis in the affected area. There was no cardiospasm. Functional recovery was observed eight to ten weeks after operation.

operation to an average of 5.6 cm. three months after operation, compared to a circumference of 3.0 cm. in a control specimen of similar length. There was one death three months after operation from chronic aspiration caused by persistent cardiospasm.

Summary.—In a group of seventeen dogs, there was one anesthetic death, one death from perforation of the esophagus, and a third from pulmonary sepsis caused by aspiration secondary to cardiospasm. Denervation caused temporary cardiospasm as well as dilatation and loss of peristalsis in the mobilized esophageal segment. Primary peristalsis returned ten weeks after operation but persistent cardiospasm delayed complete functional recovery until twelve to twenty-six weeks after denervation.

Group IV.—In this group of sixteen dogs, esophageal mobilization extended from a point 2.0 cm. above the aortic arch, through the diaphragm, and along the greater and lesser curvatures of the stomach for 3.0 cm. Vessels divided included the lower esophageal branches of the subclavian artery, the bronchoesophageal, aortic, phrenic, and left gastric arteries, and the upper vasa brevia. Denervation compromised the entire vagal supply to the devascularized area (Fig. 1).

There was one anesthetic death. There were no deaths attributable to the devascularization and denervation in this group. Four animals were sacrificed to obtain injection specimens during the first week after operation. Four subjects died of distemper within three weeks of operation. One died of intussusception during the sixth postoperative week. Six dogs were studied twenty weeks or longer for functional recovery.

Four dogs began regurgitating food, and three of these saliva, two to four weeks after operation, and continued to do so for four to twelve weeks. However, no subject showed evidence of malnutrition or chronic aspiration.

All the dogs exhibited nearly uniform roentgenologic findings. Two days after operation peristalsis and segmentation were absent in the thoracic esophagus of two dogs. The contrast medium was ingested, swallowed, and carried by normal primary peristalsis to the thoracic esophagus, which would distend markedly until the dog either stopped eating or regurgitated. Eventually the esophagus emptied, usually after at least ten minutes. Spasm of the cardiac sphincter was more prolonged than in any of the other groups, and extremely active peristalsis in the upper esophagus was necessary to overcome this spasm. Although weak peristalsis was seen occasionally in all dogs fluoroscoped two to ten weeks after operation, moderately strong peristalsis was not present until twelve to fourteen weeks. At this time, cardiospasm was still too severe to allow prompt complete emptying of the esophagus.

Adequate functional recovery was characterized by active primary peristalsis, minimal dilatation of the lower eighth of the thoracic esophagus, and minimal cardiospasm. If food were given continuously, it would pass down the esophagus with a brief delay at the cardia. The terminal portion of the food bolus remained temporarily in the lower end of the thoracic esophagus. This phenomenon was observed in three long-term survivors twenty to twenty-nine weeks after operation. One dog was sacrificed during the twenty-eighth postoperative week because cardiospasm persisted and primary peristalsis had not reappeared in the thoracic esophagus. Two long-term survivors showed no discernible abnormalities thirty-two and thirty-four weeks after operation. Cardiospasm, gradually decreasing over a period of twenty-five to thirty-four weeks, appeared to be the disturbing factor in functional recovery.

Maximal dilatation of the thoracic esophagus measured in the roentgen films was 4.6 cm. and occurred usually thirteen weeks after operation. This coincided with the first fluoroscopic evidence of primary peristaltic return (Fig. 6). Tonus began to reappear at approximately ten weeks, and segmentation at five.

There were no deaths from insufficiency of the esophageal blood supply. Small mucosal ulcerations in the lower thoracic esophagus of three subjects, sacrificed at a date later than that at which perforations occurred in other groups, were, therefore, considered to be incidental findings.

Neuropathologic changes were suggested by the dilatation of the thoracic esophagus in seven of the twelve specimens examined two days to thirty-two weeks after denervation. The dilated esophagi ranged in circumference from 3.5 to 5.0 cm. The latter was obtained three weeks after operation.

The dissection mobilizing the cervical esophagus from the thoracic inlet to the cricoid cartilage, including the posterior and lateral surfaces of the laryngeal portion, interrupted the afferent and efferent fibers in this segment. The interruption over so wide an area of the continuity of the reflex arcs responsible for the latter part of the pharyngeal and all of the esophageal stages of deglutition (that is, Groups I and VI) accounts for the absence of peristaltic

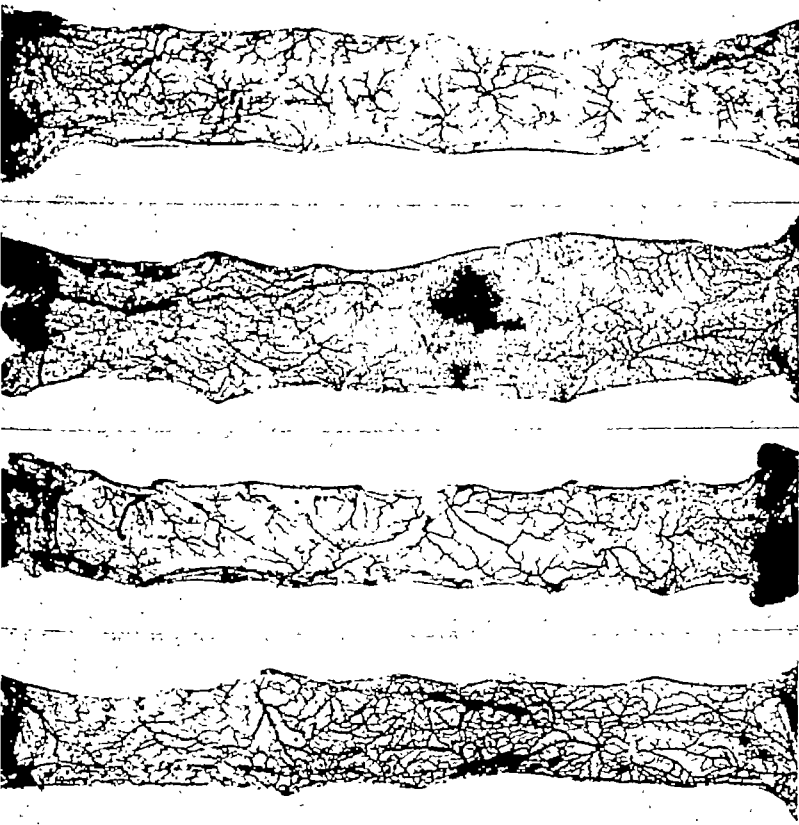


Fig. 7.—Representative specimens of esophagi with arterial blood supply injected. *A*, Normal control; note segmental arrangement of blood supply. *B*, Group I. Esophagus mobilized from cricoid cartilage to diaphragm. Note dilated longitudinal arteries extending into ecchymotic area. *C*, Group III. Esophagus mobilized from a point 2.0 cm. above the aortic arch to the diaphragm. Note dilated longitudinal arteries in the inferior half of the specimen. *D*, Group IV. Esophagus mobilized from a point 2.0 cm. above the aortic arch, through the diaphragm, to the cardia. Blood vessels of the greater and lesser curvatures ligated and divided, 2.0 cm. beyond the cardia.

Group VI.—In this group of five dogs, mobilization of the upper cervical esophagus eliminated the esophageal branches of the thyroid artery. Thorough denervation affected efferent and afferent fibers of the glossopharyngeal and vagus nerves (Fig. 1).

There were no deaths from stricture, perforation, or aspiration. One dog died of distemper eight weeks after operation.

Clinically, these dogs showed no evidence of either aspiration or malnutrition. None regurgitated either saliva or ingested fluids.

During fluoroscopy the contrast medium was ingested, and with normal deglutition it entered the esophagus. It then dilated the inert upper esophagus until the contents sank to the unaffected portion, which emptied rapidly by primary peristalsis. All subjects showed temporary dilatation and loss of primary peristalsis in the denervated area, but no cardiospasm. Peristalsis returned and dilatation disappeared four to eight weeks after operation in three of the four long-term survivors. The fourth subject showed normal peristaltic activity at twelve weeks.

Summary.—There were no perforations, strictures, or other lesions indicating circulatory insufficiency in this group. Denervation caused local loss of primary peristalsis, but no cardiospasm. Complete recovery occurred, on the average, six weeks after operation.

DISCUSSION

Throughout the experiments we were impressed by the richness of the extensive longitudinal intrinsic arterial plexus demonstrated in the injected specimens (Fig. 7). We feel that these anastomosing intrinsic arteries were responsible for maintaining esophageal viability after localized portions of the esophagus had been denervated and devascularized.

In this series of sixty-three mobilizations of various portions of the esophagus in dogs we considered perforation and stricture to be evidence of serious vascular insufficiency. Mucosal ulceration, observed in several specimens in Groups III and IV subsequent to the expected time of perforation, presumably had little effect on either the viability or the function of the esophagus.

From our data (Figs. 1 and 2) we learned that either the cervical (that is, Group VI) or the thoracic esophagus from the cardia to a point about 2.0 cm. above the aortic arch (Groups III and IV) could be mobilized with safety. Devascularization of both the thoracic and cervical portions of the esophagus (that is, Groups I and II) caused serious vascular lesions in almost one-half of the animals. We found that no specific artery was vital to the esophageal circulation, nor was any one area more susceptible than another to necrosis after devascularization. Perforations were usually found in the midpoint of a devascularized segment.

Before considering the effects of denervation it would be well to summarize the swallowing mechanism. Normal deglutition consists of a series of coordinated reflexes mediated by extrinsic nerves from medullary and pontine centers. The *afferent* or sensory fibers of the reflexes are provided by the branches of the glossopharyngeal and trigeminal nerves, and by the pharyngeal and superior laryngeal branches of the vagus. These branches innervate the mucosa near the entrance to the pharynx. The *efferent* or motor fibers of the reflexes are supplied by the hypoglossal nerve to the tongue muscles, the pharyngeal branches of the glossopharyngeal nerve, and the pharyngeal and esophageal fibers of the vagus, to the muscles of the pharynx and esophagus, and the third division of the trigeminal nerve to the mylohyoid.

It is evident from these experiments that a considerable portion of the esophagus can be separated from its regional blood supply with much less chance of serious complications than has been previously accepted. Surgical mobilization of one-half the entire esophagus is a safe procedure. Groups III and IV indicate that most of the thoracic esophagus can be freed even down to the stomach without compromising the blood supply. Group VI shows that the entire cervical esophagus can be freed without impairing its blood supply. To free the entire esophagus (cervical and thoracic) results in a very high incidence of vascular failure, as demonstrated in Groups I and II.

It should be emphasized that if a portion of the esophagus is to be resected, a proportionately larger segment of the esophagus may be freed from its vascular supply, since the area within the poorest circulation will be removed.⁶

It is clear from these experiments that after separating the esophagus from its bed, changes in function are observed which are caused by injury to the nerve supply. This division of nervous pathways for the most part results in a loss of peristalsis, which, fortunately, is only temporary. When the dissection is extended beyond the diaphragm there is prolonged cardiospasm unless the vagus nerves are carefully identified and preserved. However, even if the vagi are divided at this level, recovery from cardiospasm takes place after twenty-eight to thirty-four weeks. The occurrence of these functional changes is extremely important to remember in dealing with postoperative patients in whom a portion of esophagus has been freed.

The data of this experiment suggest solutions to several problems of esophageal surgery. A short benign stricture could be resected and repaired by end-to-end anastomosis. This has been reported by Gross,¹⁸ Swenson.²⁰ Others have corrected atresia by mobilizing both segments of the esophagus extensively and performing an end-to-end anastomosis. Congenital short esophagus with diaphragmatic hernia has been successfully treated by mobilization of the esophagus upward to the aortic arch and downward through the diaphragm (as in Group IV), but leaving the vagus fibers intact, replacing the stomach in the peritoneal cavity, securing the esophagus in normal position with interrupted sutures, and repairing the diaphragmatic defect.²¹ The satisfactory resection of esophageal malignancy and restoration of continuity with end-to-end anastomosis would depend on the presence of an early, small, well-localized lesion. Rienhoff²² will soon report his experiences with this problem.

CONCLUSIONS

1. The esophageal blood supply is sufficiently rich to permit mobilization of large portions of the esophagus.
2. The cervical esophagus can be safely freed from its bed.
3. The thoracic esophagus can be safely mobilized from the aortic arch through the diaphragm without jeopardizing its blood supply.
4. When both the cervical and thoracic portions of the esophagus are mobilized, perforation or stricture occurs in nearly one-half of the animals.

response to swallowing in the denervated area. With normal primary peristalsis and no cardiospasm in the thoracic esophagus, and with thorough denervation of the lower pharyngeal and upper esophageal region, there was no aspiration (Group VI). With a similar, but more widespread denervation (Group I) there was cardiospasm and loss of peristalsis over the denervated area. We attribute the high incidence of fatal aspiration in Group I to cardiospasm and loss of peristalsis, and not to dissociation of pharyngeal and esophageal function.

The most fundamental concept of esophageal peristalsis is expressed in Starling's law: "Excitation of the gut at any point elicits contraction above and relaxation below the point of stimulation." Cannon¹⁵ has postulated that this is a local phenomenon mediated by the myenteric plexus intrinsic to the esophagus. Transmission of primary peristalsis, except in the lower thoracic esophagus where smooth muscle predominated, is normally dependent upon the integrity of the vagus nerves. Simple division of the esophagus without vagal interruption does not stop peristalsis, while division of the esophageal vagus fibers results in complete temporary paralysis and dilatation of the upper two-thirds of the entire esophagus. In cases of vagal interruption the myenteric plexus assumes control after about seven days and recovery of peristalsis gradually follows.¹⁴

Although the extensive interruption of the esophageal fibers of the vagus in the various groups of dogs is adequate to explain temporary loss of peristalsis, it should be noted that all of the operations were accompanied by extensive devascularization, as well. This might well be expected to damage the cells of the myenteric plexus, or even the muscle fibers themselves. In this respect the work of Cannon and Burket¹⁷ is pertinent. They demonstrated that the cells of the myenteric plexus of the intestine had great resistance to anoxemia, and could recover normal function even when absolutely deprived of their blood supply for periods up to seven hours. For this reason, and the fact that vagal interruption alone can account for peristaltic loss, we feel that partial removal of the esophageal blood supply was not the primary cause of this phenomenon, although it may have contributed to the long delay before recovery begins.

Cardiospasm, as well as changes in primary peristalsis, was observed by Cannon¹⁴ and Milhiet¹⁸ after division of the vagus below the origin of the recurrent laryngeal nerve. We noted, first, cardiospasm of varying severity in Groups I, II, III, and IV, but none in Groups V and VI. We also observed recovery from cardiospasm concomitant with the return of primary peristalsis in Group II at approximately fifteen weeks, but not in Groups III and IV until approximately twenty-one and twenty-six weeks, respectively. From our first finding we deduce that the cardiospasm noted resulted from a local and not a widespread disturbance of the esophageal innervation. From our second we learn that, although primary peristalsis reappeared almost simultaneously in Groups II, III, and IV, persistent cardiospasm greatly increased the average time required for functional recovery in the latter two groups.

BRONCHOSPIROMETRIC STUDIES IN BRONCHIECTASIS, BEFORE AND AFTER LOBECTOMY

JOAN H. LONG, M.D., CHARLES M. NORRIS, M.D., W. EMORY BURNETT, M.D.,
AND MARY R. WESTER, M.S.
PHILADELPHIA, PA.

IN RECENT years the work of Courmand and Richards¹ and Pinner and associates² has changed the concept of the measurement of pulmonary function from that of a static measurement of vital capacity to the dynamic measurement of the many aspects of respiration including the various divisions of the lung volume, ventilation, and the diffusion and transport of the respiratory gases. The present study is concerned with the limited aspect of ventilation and oxygen uptake in a group of patients with bronchiectasis and the changes in these two functions following the surgical resection of diseased lung tissue.

MATERIAL AND METHODS

The over-all pulmonary ventilation was determined in 64 patients with bronchiectasis in whom the pulmonary disease had been demonstrated and delineated by Lipiodol bronchography of all the lobes. These patients ranged in age from 14 to 57 years. There were 33 male and 31 female patients. The results of the over-all function studies were compared with the over-all function obtained by the same method on 63 normal individuals within the same age range. In 44 of these patients lobectomy was carried out, and the over-all function was measured again postoperatively, usually one to three weeks after operation, and, in the majority of cases, again several months or even years later. In 30 of the group with bronchiectasis, bronchospirometric studies were carried out; in 21 of these preoperative and postoperative studies were made several months after operation. Five other patients were studied similarly by external spirometry and bronchospirometry before and after operation. These underwent lobectomy for bronchial adenoma (3), bronchial carcinoma (1), and pulmonary cyst (1).

External spirometry measuring over-all pulmonary ventilation was carried out with the patient in the basal condition using a Roth-Benedict water spirometer. With the patient resting in the supine position a record of quiet respiration was obtained over a four to five minute period; the resulting spirogram was used for analysis of the oxygen absorbed per minute, the respiratory rate, the depth of respiration (tidal air), and the average minute volume of respiration (M.V.R.), that is, the respiratory rate times the tidal air. The soda lime canister was then removed from the spirometer to lessen the resistance³ and the patient was asked to breathe as deeply and as fast as possible for thirty seconds. A short recovery period was always recorded. From this record the maximum breathing capacity (M.B.C.) was calculated in terms of liters per minute. This method of measuring voluntary M.B.C. with the patient in the supine position gives consistently lower values than those obtained by Courmand² with the subject in the erect position. The breathing reserve at rest was derived (M.B.C. - M.V.R.) and the per cent breathing reserve calculated according to the formula $\frac{(M.B.C. - M.V.R.)}{M.B.C.} \times 100$.

A Gebauer twin spirometer was used in the bronchospirometric determinations. Although a Zavod catheter was used in a few of the early determinations, a Norris type catheter⁴ was used in the majority of the cases. The patients were not in a basal condition for this procedure. Local pontocaine anesthesia and fluoroscopic guidance were used, and the catheter was put in place with its tip in the left main bronchus.

From the Departments of Surgery and Laryngology and Broncho-Esophagology, Temple University Hospital and Medical School.

Received for publication March 20, 1949.

5. The more extensively the esophageal fibers of the vagus are disturbed near the diaphragm, the more marked is the resultant cardiospasm.

6. Denervation of the esophagus causes loss of peristalsis followed by complete recovery from six to sixteen weeks after operation.

Acknowledgment.—This work was greatly assisted by the generous cooperation of Dr. Orville T. Bailey, Dr. Jonathan Cohen, Dr. Sidney Farber, Dr. Thomas Gucker, III, Mr. Ferdinand Harding, and Dr. Edward Neuhauser, all of The Children's Medical Center, and Dr. William Richardson, formerly of this hospital.

REFERENCES

1. Carrington, G. L.: Experimental Surgery of the Esophagus, Some Factors and End-results, *Ann. Surg.* 86: 505-514, 1927.
2. Adams, W. E., Escadero, L., Aronsohn, H. G., and Shaw, M. M.: Resection of the Esophagus, a Clinical and Experimental Study, *J. THORACIC SURG.* 7: 605-620, 1928.
3. Ochsner, A., and DeBakey, M.: Surgical Aspects of Carcinoma of the Esophagus. A Review of the Literature and Report of Four Cases, *J. THORACIC SURG.* 10: 401-445, 1941.
4. Saint, J. H.: Experimental Surgery of the Esophagus, *Proc. Staff Meet., Mayo Clin.* 3: 223-224, 1925.
5. Saint, J. H.: Surgery of the Esophagus, *Arch. Surg.* 19: 53-128, 1929.
6. Swenson, O., and Clatworthy, H. W.: Partial Esophagectomy With End-to-end Anastomosis in the Posterior Mediastinum, *Surgery* 25: 839-848, 1949.
7. de Francesco, F.: Recherche experimentale sul "Blocco Chimico e Chirurgico" del Tratto Esophageo Sopracardiale e Sotto Diaframmatico, *Clin. chir.* 10: 558-616, 1934.
8. Miller, R. T., Jr., and Andrus, W. de W.: Experimental Surgery of the Thoracic Esophagus, *Bull Johns Hopkins Hosp.* 34: 109-115, 1925.
9. Ochsner, A., and Owens, N.: Antethoracic Esophagoplasty, *Ann. Surg.* 100: 1055-1091, 1931.
10. Fischer, H.: Surgical Treatment of the Esophagus, *Arch. Surg.* 6: 256-269, 1923.
11. Demel, R.: Arteries Supplying the Esophagus, *Arch. f. klin. Chir.* 128: 453-504, 1924.
12. Best, C. H., and Taylor, N. B.: *The Physiological Basis of Medical Practice*, ed. 4, Baltimore, 1947, Williams & Wilkins Company, p. 479.
13. Harding, F. R.: Unpublished data.
14. Cannon, W. B.: Esophageal Peristalsis After Bilateral Vagotomy, *Am. J. Physiol.* 19: 436-444, 1907.
15. Cannon, W. B.: Peristalsis, Segmentation, and the Myenteric Reflex, *Am. J. Physiol.* 30: 114-128, 1912.
16. Templeton, F. E.: *X-ray Examination of the Stomach*, Chicago, 1944, University of Chicago Press, p. 91.
17. Cannon, W. B., and Burket, I. R.: The Endurance of Anemia by the Cells of the Myenteric Plexus, *Am. J. Physiol.* 32: 347, 1913.
18. Milhiet, H.: Reflexions sur la mega esophage: A propos d'un cas traite par l'intervention chirurgicale et a propos dilatations paralytiques experimentales de l'esophage du chien, *Presse med.* 47: 1639-1641, 1939.
19. Gross, R. E.: Treatment of Short Stricture of the Esophagus by Partial Esophagectomy and End-to-End Esophageal Reconstruction, *Surgery* 23: 735-744, 1948.
20. Swenson, O.: End-to-end Anastomosis of the Esophagus for Esophageal Atresia, *Surgery* 22: 324-334, 1947.
21. Gross, R. E.: Unpublished data.
22. Rienhof, R.: Personal communication.

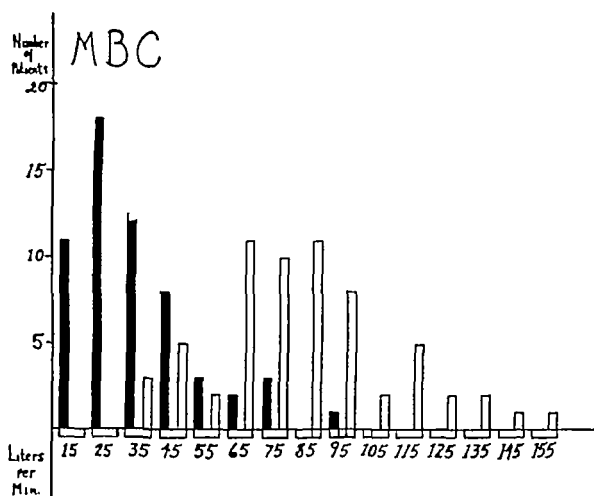


Fig. 1.—Maximum breathing capacities (black bars) obtained by maximum voluntary effort in 61 patients with bronchiectasis compared with maximum breathing capacities (white bars) obtained in 63 normal subjects in the same age group.

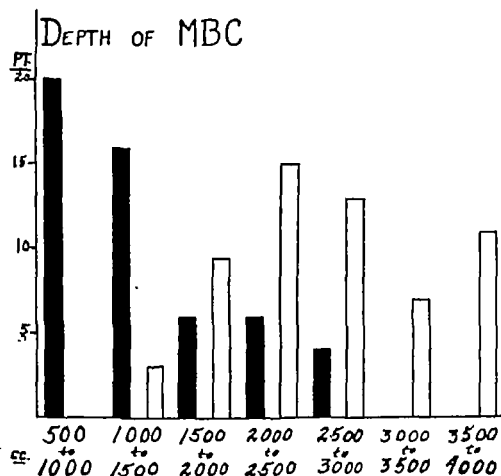


Fig. 2.—Depth of respiration (black bars) achieved during maximum voluntary effort by 64 patients with bronchiectasis compared with the depth of respiration (white bars) achieved by 63 normal subjects in the same age group.

The division of function between the two lungs was calculated from the broncho-spirometric record with reference to two factors: the per cent of the total oxygen absorbed and the per cent of the total volume of air moved by each lung. By referring these relative functions back to the resting oxygen absorption and tidal volume as determined by external spirometry under basal conditions, the probable resting function of each lung was estimated.

THE EFFECT OF BRONCHIECTASIS ON CERTAIN ASPECTS OF PULMONARY FUNCTION

Bronchiectasis is a chronic disease of the bronchi characterized by a cough usually productive of large amounts of purulent sputum. Consideration of the pathologic anatomy leads to an understanding of the physiologic changes which may occur with this disease. The affected bronchi are abnormally dilated and filled with mucopurulent secretion. The lesion of the bronchial walls may range from moderate dilatation with mild infection to enormous dilatation with loss of cilia, elastic tissue, and smooth muscle, and at times replacement of the walls by fibrous tissue^{5, 6} with severe infection. Some part of the lung parenchyma is also usually involved by pneumonitis, hemorrhage, atelectasis, or fibrosis and scarring. Both ventilation and diffusion of respiratory gases to and from the blood may be impaired. The movement of air to and from the alveoli may be limited by the loss of elastic and smooth muscle tissue or frankly blocked by the accumulated secretions. The passage of respiratory gases to and from the blood may be further impaired by involvement of the alveoli in the disease process. Functional studies give some indication of the extent to which the normal pulmonary physiology is altered by these pathologic changes.

In this group of 64 patients with bronchiectasis there was little objective evidence obtained from external spirometry tracings alone that the respiration at rest was other than normal. In the majority of the cases the respiratory rate was less than 20 per minute, or within normal limits; in 11 it ranged between 20 and 24, and in 1 it was recorded as 30. Three patients who had widespread bronchiectasis complained of dyspnea at rest despite an apparently "normal" resting ventilation as recorded by external spirometry. Arterial oxygen content determinations would have contributed to a better understanding of the state of the resting pulmonary function.

The maximum ventilatory ability as measured by the voluntary maximum breathing capacity (M.B.C.) was frequently below normal (Fig. 1). To a certain extent it might be stated that the more extensive the disease the lower the maximum breathing capacity. The limited depth of respiration during maximum effort was largely responsible for the lower maximum breathing capacity values (Fig. 2). The rate and depth of respiration during maximum effort depend not only upon the patency of the airways but also upon the muscular strength of the individual and his ability to sustain muscular effort, which in turn are related to his general condition, his nutritional status, and the presence or absence of infection. Occasionally a striking increase in the maximum breathing capacity followed vigorous treatment which reduced infection and the amount of mucopurulent secretions and improved the general condition of the patient. Such treatment included bed rest, abolition of smoking, postural and bronchoscopic drainage, chemo- and antibiotic therapy, supplementary protein in the diet, iron

per cent division of the total lung volume by weight among the various lobes and their subdivisions. From these values the theoretical function which might be expected to remain in lung segments remaining on the operated side after lobectomy was calculated. This assumes, of course, that function parallels anatomic size, which is not necessarily true, particularly in an abnormal lung.

Two-thirds of the cases showed that the diseased lung ventilated a greater volume of air than might be anticipated on the basis of complete lack of function of the bronchiectatic areas. One-half of the total number showed a greater oxygen absorption from the diseased lung than predicted. From this it would appear that either the diseased segments were not completely functionless or that the undiseased portions had compensated for the loss of function to some extent. Both situations probably occur.

In summary, the presence of bronchiectasis in one or two lobes does not usually impair the resting ventilation appreciably. It does decrease the maximum breathing capacity below normal levels, particularly impairing the depth of respiration achieved, in a manner roughly proportional to the extent of the disease. The function of oxygen absorption is frequently more impaired by bronchiectasis than the function of ventilation. The divisions of both functions between the two lungs may not differ from the normal if the disease process is limited to only a few bronchopulmonary segments. It must be remembered that bronchspirometry measures only the relative functioning of the two lungs. We are not able to say with certainty how much adjustment has taken place in the undiseased portions of lung although compensatory increases in ventilation and oxygen absorption probably occur.

CHANGES IN PULMONARY FUNCTION FOLLOWING REMOVAL OF DISEASED SEGMENTS OF LUNG TISSUE

Questions then arise as to what occurs when a diseased segment or lobe of a lung is removed. Can an improvement in the over-all pulmonary function be expected with the removal of infected and distorted lung tissue, and what may happen to the function of the lobes of lung remaining on the operative side?

Lindskog⁵ in 1939 stated that the removal of one or two lobes of a lung caused no permanent reduction of pulmonary volume, using the vital capacity and its component parts as criteria. Birath⁶ studied both lobectomy and pneumonectomy patients two to thirteen years after operation and reported that the total lung volume was less reduced than the corresponding lung parenchyma, suggesting that distention of remaining lung tissue occurred postoperatively. The total dead space was found to be increased, probably due to the increased inflation of the lung occurring with distention of the parenchyma. Kay, Meade, and Hughes⁷ concluded from 26 bronchspirometric studies carried out following lobectomy that the function of the remaining pulmonary tissue on the operated side was dependent largely on the postoperative pleural complications and that the postoperative function on the operated side in a patient with an uneventful convalescence may be within normal limits.

administration, and blood transfusions. One patient with bilateral bronchiectasis had an initial maximum breathing capacity of 30 liters per minute; after one month of treatment this was increased to 66 liters per minute. A child with bilateral bronchiectasis doubled the depth of respiration on maximum effort after two weeks of intensive therapy. A fifteen-minute period of postural drainage alone was found to increase one patient's vital capacity by 400 c.c. (from 1,500 to 1,900 c.c.). On the other hand there was no change in the maximum breathing capacity of another patient after one month of similar intensive therapy.

External spirometry gives information only about the total lung function, and more specifically, only about total ventilatory function. Bronchosprometry, which separates the air moved by the right and left lungs simultaneously, provides the means of studying more closely the changes both in ventilation and in oxygen absorption occurring in pulmonary tissue diseased by bronchiectasis. It must be remembered that these studies will not necessarily give an exact picture of what is occurring in a diseased lung segment for it is not yet possible to measure the function of one segment alone. Bronchosprometry affords only a comparison of one lung with the other, a comparison of one lung containing diseased segments with the opposite lung which may be normal or partly diseased. At best it is not a question of comparing normal lung with abnormal lung but rather a problem of comparing normal lung with a combination of normal and abnormal lung.

Bronchosprometry was carried out on 39 patients with bronchiectasis. In 22 of these patients the bronchiectasis was essentially unilateral. In over one-half (12) of the cases of unilateral bronchiectasis the oxygen absorption from the diseased lung was impaired to a greater extent than the ventilation of air by this lung, that is, the per cent of the total oxygen uptake was less than the per cent of the total air ventilated. Of the remainder of the cases (10) the right:left ratios of oxygen absorption and ventilation were within normal limits in 4; oxygen absorption and ventilation were equally impaired in 2; while in 4 others the ventilation was reduced more than the oxygen absorption. We are unable to tell from these measurements how much the normal portion of lung on the affected side compensated in volume or in oxygen uptake for the diseased portion. At the moment it can be said that the data appear to suggest that in unilateral bronchiectasis oxygen absorption usually tends to be impaired to a greater extent than ventilation. That is, in the diseased lung the movement of air is less noticeably affected than the gaseous exchange across the alveolar membrane. Consideration of the pathologic anatomy alone might lead one to expect this to be the case. These results substantiate the work of Kay, Meade, and Hughes,⁷ who likewise found that in bronchiectasis the uptake of oxygen from the diseased lung was generally proportionally less than the ventilation through that lung.

A comparison was made in the 22 cases of unilateral bronchiectasis between the function of the diseased lung and the function which might be expected if the diseased portion were completely functionless. A preliminary anatomic study was carried out on two pairs of lungs and an estimate was made of the

24. LLL, Lingula	540	700	600	3.8	7.0	4.8	37.8	28.8	64.5	1350	800	1000	Fever atelectasis (17 days)
25. LLL, Lingula	650	600	425	7.8	6.6	5.5	25.5	33.6	27.3	1500	1050	1050	Fever (11 days)
26. LLL, Lingula	440	260	400	9.7	9.4	6.0	27.0	11.2	20.8	775	435	1025	None
27. LLL, Lingula	350	220	580	8.1	7.3	8.1	21.0	12.2	18.5	700	1500	2800	Atelectasis (11 days)
28. RML, RLL	650	800	750	7.6	9.6	0.0	63.8	69.0	84.0	2000	750	750	Atelectasis (8 days)
29. RML, RLL	300	350	400	6.0	6.7	8.0	28.3	28.5	37.5	2300	1350	2400	Fever (13 days)
30. LLL, LUL, partial	1000	750	650	8.0	11.2	7.8	73.5	45.8	76.8	500	800	1100	Fever (7 days)
31. LLL (bilateral disease)	400	400	400	7.2	7.2	14.3	56.0	35.2	35.0	1750	800	580	Fever (9 days)
32. RLL partial (bilateral disease)	600	400	650	7.2	7.2	14.3	17.0	16.0	11.6	850	800	700	None
33. RUL partial (bilateral disease)	600	575	450	8.4	6.9	5.4	18.5	18.4	14.3	500	575	850	None
34. LLL partial, Lingula	650	430	430	13.7	10.6	9.0	14.7	25.2	21.0	1250	900	600	None
35. Following LLL (bilateral disease), RML, RLL	480	460	650	6.9	6.5	0.0	37.5						
36. Lingula	640	500	750	6.4	6.5	0.0							
37. LLL, partial	360	400	400	7.9	10.8	8.0	32.5	38.4	53.2	650	800	900	None
38. LLL	400	300	600	8.0	9.0	12.0	53.2	52.2	51.6	900	900	1175	None
39. LLL	500	600	600	6.3	4.2	4.2	33.6		30.0	1400	1500	1500	Fever (4 weeks)
40. LLL, Lingula	350	350	460	4.5	9.2	9.2	32.2		58.1	700	1650	1650	None
41. LLL, Lingula	380	500	500	10.6	12.0	12.0	14.4		19.2	800	1200	1200	None
42. LLL, Lingula	500	500	500	8.0	7.0	23.8	23.8		42.0	625	1000	1150	None
43. LLL partial, Lingula	375	380	500	0.4	8.0	12.5	30.0	22.4	41.4	1500	800	1150	None
44. LUL	608	400	754	6.7	8.3	8.3	55.6		27.0	1500	600	1600	Empyema Atelectasis (8 days)
45. RML, RLL partial	550	400	450	9.4	8.8	5.9	36.0	16.2	54.4	1500	900	1500	Fever (11 days)
46. RLL	700	650	950	7.7	10.4	6.6	33.0	28.8	42.0	1000	425	950	None
47. RLL (following LLL bilateral disease)	400	500	700	9.9	6.0	11.4	30.0	17.0	41.8	1050	750	700	None
48. RML	540	400	500	7.2	12.0	10.8	39.6	25.5	16.8	700	725	1050	None
49. RUL	650	400	500	7.0	5.2	6.0	22.4	27.6	39.0	2000	1050	1650	None
			500	9.1	9.5	9.5	32.3		30.6	950		900	None

TABLE I. DATA OBTAINED PRE- AND POSTOPERATIVELY BY EXTERNAL SPIROMETRY ON FORTY-NINE PATIENTS WHO UNDERWENT PULMONARY RESECTION

RESECTED SEGMENTS	TIDAL VOLUME			MINUTE VOLUME			MAXIMUM BREATHING CAPACITY			DEPTH REACHED IN M.B.C.			COMPLICATIONS
	PREOP. (C.C.)	2 WEEKS POSTOP. (C.C.)	LATER POSTOP. (C.C.)	PREOP. (LIT- ERS)	2 WEEKS POSTOP. (LIT- ERS)	LATER POSTOP. (LIT- ERS)	PREOP. (LIT- ERS)	2 WEEKS POSTOP. (LIT- ERS)	LATER POSTOP. (LIT- ERS)	PREOP. (C.C.)	2 WEEKS POSTOP. (C.C.)	LATER POSTOP. (C.C.)	
1. RLL partial	900	700	1250	8.1	8.4	12.5	70.0	57.0	101.0	2500	1350	2200	None
2. LUL	520	500	475	6.8	9.5	6.7	78.3	59.5	75.0	1400	850	1500	None
3. LLL	700	600		7.0	9.6		37.4	22.8		1700	950		
4. LLL	550		500	8.3		9.5	14.4		19.2	1200		800	None
6. LLL and lingula	550	450	850	11.0	9.5	9.4	32.0	17.0	35.2	1000	500	1100	None
7. LUL and LLL partial	500		400	6.0		5.6	16.2		19.2	900		800	TB
8. RLL, RML	475	600	620	7.1	7.2	7.5	44.0		31.5	1100	810	1050	Empyema
9. RLL, RML	700	560		7.7	10.6		48.0	28.8		1200	600		None
10. Lingula (bilateral disease)	800			10.0			69.0			1500			None
11. LLL (bilateral disease)	400	400	625	7.2	6.4	9.4	17.2		18.9	1075	1000	1350	Fever
12. RLL partial	650		600	13.0		10.2	41.8		63.7	1900		2450	Fever
13. Lingula	880		1100	12.3		7.7	23.5		58.0	1175		2900	(9 days)
14. Lingula	900	700	600	15.3	16.8	7.2	44.0	40.0	39.1	1100	950	1150	None
15. RML	500		630	9.0		11.3	78.2		56.0	2300		2000	Fever
16. LLL partial	300		550	5.4		8.8	36.4		30.8	1300		1100	(8 days)
17. LLL partial	750	500	660	9.7	9.5	5.9	52.0	33.6	52.8	2000	1200	2400	None
18. LLL	400	275	500	5.6	4.7	6.5	22.0	11.7	26.0	1000	650	1000	Atelectasis
19. LLL	700		700	6.3		7.0	20.6		30.0	1030		1500	(5 days)
													(6 days)
													(11 days)
20. LLL	650	500	460	11.1	12.0	11.1	48.4	26.4	45.6	1500	1200	1200	atelectasis
21. LLL	750	1000	590	6.0	9.0	8.3	28.0	24.2	40.6	2000	1100	1450	Empyema
22. LLL	375	480	450	6.0	8.2	6.3	20.8	14.0	23.0	650	500	500	Fever
22. Lingula	450	450	300	6.3	7.6	5.4	23.0	19.0	18.9	500	500	450	Empyema
23. RUL	1050	1250	1500	7.5	10.0	12.0	84.0	41.6	62.0	3500	1600	2200	None

closely. There are three possibilities with respect to the function of the remaining lung tissue: (1) No change occurs in the ratios of the air moved or the oxygen absorbed by the two lungs, in which case any compensatory changes or increases in volume or in oxygen uptake are contributed to equally by both the operated and unoperated sides; (2) the per cent of the total ventilation and oxygen absorption of the operated side is reduced with the result that compensatory changes occur largely in the unoperated lung; (3) there is an increase in the per cent of the total function contributed by the remaining segment of lung on the operated side, which means that it is this segment which is providing a large part of the compensation.

A comparison of the percentage distribution of the movement of air and the oxygen uptake as determined by bronchspirometry in 24 cases pre- and postoperatively showed that all three of the aforementioned possibilities occurred, but with different frequencies (Table II). Moreover, the changes in ventilation and in oxygen absorption did not parallel each other in every case.

In 10 cases the per cent of the total volume of air moved by the operated lung was significantly decreased postoperatively, which can only mean that any over-all increases in the depth of ventilation which occurred were provided by the unoperated lung. In 10 cases there was no change in the ratios of the volume of air ventilated by each lung. This means that either the resected lobe had been completely without function prior to operation or that the remaining lung segments on the operated side assumed the function formerly contributed by the diseased lung tissue. This does not mean that the ventilatory function of the operated lung had become normal, but merely that the division of function between the two lungs was the same as it had been preoperatively. Very infrequently (in 4 cases) there was an actual increase in the proportion of air moved by the operated lung.

The findings concerning the ratio of the oxygen absorbed by the two lungs were somewhat different and did not necessarily parallel the changes in ventilation. In 12 cases there was a decrease in the per cent of oxygen absorbed by the operated side, in 6 cases there was an increase, and in 6 cases there was no change.

Of the 24 patients on whom bronchspirometry was done before and after resection of diseased lung tissue, 18 had some reduction either in the per cent of the oxygen uptake or per cent of total ventilation contributed by the operated lung. Four of the 18 had a reduction in both functions. Six of the 24 patients had either no change or an increase in the functions of ventilation and oxygen absorption of the operated lung. The findings in the 14 patients who had had partial lobectomies or a lobectomy plus resection of a segment of a second lobe did not differ from those of the entire group.

Certain other facts might also be pointed out. After the surgical removal of the diseased lung tissue, the ratios of the air moved by each lung and the oxygen absorbed did not usually return to normal unless they were normal preoperatively. In 4 cases only was the resulting ratio of the ventilation by the right and left lung normal (right 53 per cent $\pm$ 5; left 47 per cent $\pm$ 5); in 3 of these the ratio had been normal preoperatively and had not been changed

In our series preoperative and postoperative studies on 49 patients were compared for changes in over-all pulmonary function following resection of portions of diseased lung tissue (no pneumonectomies were included in this study) (Table I). The patients ranged in age from 14 to 57 years at the time of operation (average, 32.7 years). Operation was carried out in 44 patients for the treatment of bronchiectasis, in 3 for bronchial adenoma, in 1 for bronchiogenic carcinoma, and in 1 for the removal of a pulmonary cyst which occupied the lingula of the left upper lobe.

In the immediate postoperative period (two weeks or less), while the patient was still in the hospital, changes were noted both in the resting function and in the function with maximum voluntary effort. The resting respiration was usually more rapid than it had been prior to operation. There was little change in the depth of respiration at rest. There was, however, a marked decrease in the depth of respiration attained during maximum effort as well as in the vital capacity. Pain with deep respiration together with a lack of movement of the diaphragm on the operated side due to crushing of the phrenic nerve at operation were undoubtedly factors which contributed to these decreases. When complete healing had taken place and the thoracic contents had become stabilized, these difficulties were obviated and the over-all ventilatory function returned to its preoperative level or increased, as will be shown.

Comparison of the postoperative studies on 44 patients studied five months or more postoperatively with the preoperative tests showed that in this later period the resting respiration had returned to its preoperative character. The oxygen absorption at rest, the rate and depth of respiration; and, hence, the minute volume were the same as they had been preoperatively. Somewhat surprising results were encountered in the comparison of the maximum breathing capacities. In more than one-half the cases there had been no decrease in the depth of respiration attained during maximum voluntary effort; in 8 cases there had been a significant increase in the depth (more than 300 c.c.). The maximum breathing capacity is the product of the rate and the depth. Constancy or the increase of the depth coupled with either no change or an increase in rate during maximum effort resulted in the following facts: not only had the maximum breathing capacity returned to the preoperative values in 15 cases, but in 19 cases it had actually increased significantly (more than 15 per cent), the increase amounting to 147 per cent in 1 instance. In only 9 cases was there a significant decrease in maximum breathing capacity. These decreases were due primarily to a decreased depth of respiration attained during the test period. Two of the patients with decreased maximum breathing capacity had had empyema during the postoperative period; one had fever for eight days; 2 others had extensive bronchiectasis on the opposite side. There was no apparent reason for the decrease in the maximum breathing capacity in the remaining 5 cases.

The fact that the resting respiration (ventilation and oxygen uptake) was the same as it had been preoperatively in all cases and that the maximum breathing capacities were the same or better in 82 per cent of the cases suggests that compensatory changes may have taken place in the remaining lung tissue. Bronchspirometric studies furnish a means of studying these changes more

by operation. In one case there had been an increase in the per cent of the air moved by the remaining segment of lung on the operated side. Kay and associates⁷ found normal ratios postoperatively in some of their patients; the number was not stated.

An attempt was made to correlate the postoperative course of these patients with the changes in the functions of oxygen uptake and ventilation of the operated lung as shown by bronchspirometry. Of the 8 patients with an absolutely uneventful postoperative course (that is, with no fever after the third postoperative day and no x-ray evidence of atelectasis after the first four to five days postoperatively), 4 had some decrease in function. All 4 showed a reduction in the per cent of total air ventilated by the operated lung, but only 2 had a reduction in the per cent of total oxygen absorption.

Six patients had x-ray evidence of atelectasis for 5, 7, 8, 11, 11, and 17 days, respectively. Of these, 3 had a reduction in the per cent of total oxygen taken up by the operated lung with no change in ventilation. One had a decrease in both of these functions. Surprisingly, 2 patients in whom the atelectasis was present for 11 and 17 days had no impairment in oxygen absorption in the remaining segment after it had re-expanded.

Of the 7 patients who had fever alone for 7 to 21 days postoperatively, with no apparent etiology other than the chest lesion, 4 showed some reduction in function of the operated lung. All 4 showed a decrease in the per cent of oxygen taken up by the remaining lung segment and only 2 had a decrease in the per cent of air moved.

The 2 patients with postoperative empyema and bronchopleural fistulas did badly. Both had very little function remaining on the operated side either with regard to oxygen absorption or ventilation of air.

Considering these same bronchspirometric findings from another viewpoint, of the 12 patients who had reductions in per cent of total oxygen use of the operated lung following resection, 9 had some postoperative complication (atelectasis, fever, or empyema); of the 10 patients with a reduction in the per cent of total ventilation by the operated lung, only 6 had some postoperative complication, 4 having what was considered to be an uneventful postoperative course. However, not every patient with postoperative complications suffered impairment of oxygen absorption or ventilatory ability. To summarize, reductions in the ventilation may occur even with an uneventful postoperative course, whereas reductions in oxygen uptake are much more likely to be associated with complications such as fever, atelectasis, or empyema. It is also possible for a patient to have such complications and to suffer no impairment in the functions of ventilation or oxygen uptake of the operated lung.

SUMMARY AND CONCLUSIONS

Two aspects of respiratory function were studied, namely, ventilation and oxygen uptake, in a group of 64 patients with bronchiectasis and 5 patients with other pulmonary disease. These were compared with 63 normal subjects.

In bronchiectasis it was found that the maximum breathing capacity, as shown by external spirometry, was reduced below normal in a manner roughly

Table II
DATA C

RESECTED LUNG SEGMENTS	OXYGEN CONSUMPTION					TIDAL VOLUME					POSTOPERATIVE COMPLICATIONS	
	PREOPERATIVE			POSTOPERATIVE		PREOPERATIVE			POSTOPERATIVE			
	RIGHT-LEFT	RATIO		RIGHT-LEFT	RATIO	RIGHT-LEFT	RATIO		RIGHT-LEFT	RATIO		
1. RLL partial	216	184	54:46			540	360	60:40				
2. LUL	149	67	69:31			260	260	50:50				
3. LLL	184	104	64:36			525	175	75:25				
4. LLL	137	113	55:45			386	164	61:39				
5. LLL, lingula	108	75	59:41			372	248	60:40				
7. LUL, part LLL	248	65	79:21			380	120	76:24				
8. RML, RLL	103	87	54:46			252	223	53:47				
9. RML, RLL	92	138	40:60			231	469	33:67				
10. Lingula (bilateral disease)	88	78	53:47			272	188	59:41				
11. LLL ((bilateral disease)		62	70:30			164	136	41:59				
12. RLL partial	87	66	57:43	158	68	70:30	351	299	54:46	312	298	52:48
13. Lingula	154	52	71:29	149	104	59:41	625	255	71:29	632	418	62:38
14. Lingula	116	84	58:42	114	102	53:47	513	387	57:43	390	210	65:35
15. RML	155	345	31:69	126	294	30:70	276	199	58:42	353	277	56:44
16. LLL, basal segments	110	110	50:50	143	40	78:22	150	150	50:50	429	121	78:22
17. LLL, basal segments	91	84	52:48	193	82	70:30	563	187	75:25	488	172	74:26
18. LLL	182	111	62:38	151	62	71:29	252	148	63:37	335	165	67:33
19. LLL	138	39	78:22	175	28	86:14	406	294	58:42	406	294	58:42
20. LLL	238	45	84:16	179	14	93:7	520	130	80:20	382	78	83:17
21. LLL	146	54	73:27	199	41	83:17	450	300	60:40	460	130	79:22
22. LLL, lingula	114	69	62:38	127	26	83:17	195	180	52:48	373	77	83:17
23. RUL	119	164	42:58	156	144	52:48	430	620	41:59	570	930	38:62
24. LLL, lingula	227	93	71:29	219	81	73:27	350	190	65:35	462	138	77:23
25. LLL, lingula	120	80	60:40	144	139	76:24	416	234	64:36	267	383	56:44
26. LLL, lingula	113	30	79:21	124	26	83:17	246	194	56:44	273	52	84:16
27. LLL, lingula	138	75	65:35	140	93	60:40	178	172	51:49	435	145	75:25
28. RML, RLL	78	182	30:70	107	136	49:51	360	590	38:62	420	330	56:44
29. RML, RLL	92	133	41:59	63	127	33:67	138	162	46:54	168	232	42:58
30. LLL, LUL, except for apical segment	158	85	65:35	222	94	70:30	580	420	58:42	428	222	66:34
31. LLL (bilateral disease)			57:43	138	62	69:31			42:58	358	132	73:27
32. RLL (bilateral disease, partial)	80	120	42:58	154	166	48:52	228	372	38:62	272	378	42:58
33. RUL partial (bilateral disease)	22	78	22:78	69	169	29:71	220	280	44:56	180	270	40:60
34. Following LLL partial RLL, RML, RUL.	190	60	70:24	175	75	70:30	288	142	67:33	253	397	39:61
35. Following LLL RML, RLL.	70	114	41:59	156	117	57:43	282	358	44:56	413	337	55:45

AN INSTRUMENT FOR PASSING LIGATURES

JAMES R. MCCORRISTON, B.A., M.D., C.M., M.Sc.

MONTREAL, CANADA

AN INSTRUMENT has recently been devised for passing ligatures about deeply placed structures. This report is for the purpose of describing the instrument.

The over-all length of the ligature passer is 10 inches (Fig. 1). It is $\frac{5}{8}$ inch in breadth near the proximal (handle) end, and it tapers to a breadth of $\frac{3}{16}$ inch near the curved distal end. The curve near the distal end is one quarter

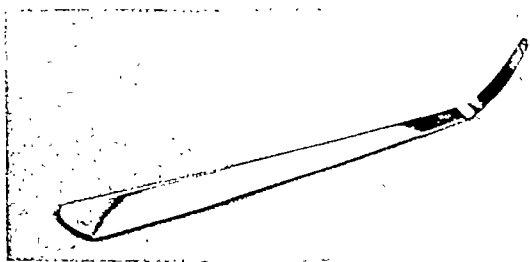


FIG. 1.

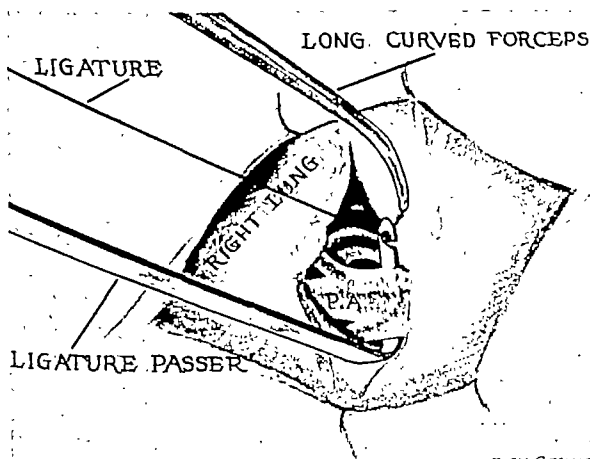


FIG. 2.

From the Research Surgical Service, The Montreal General Hospital.
Received for publication July 18, 1949.

proportional to the extent of the disease. This reduction was due in the main to a decrease in the depth of respiration attained during maximum voluntary effort.

Bronchspirometric studies showed that in bronchiectasis the oxygen uptake in the diseased lung was often more reduced than the volume of air moved. The reductions in these functions were usually less than would be predicted from anatomic studies if the diseased lung were completely functionless, indicating that there was probably both some residual function in the diseased portions and some compensation on the part of the undiseased segments.

Although the reduction in the per cent of air moved on the diseased side was made up, in part, by an increase in the per cent of the total air moved by the undiseased lung, the over-all ventilation was normal only during quiet respiration. It was reduced during effort.

Lobectomy or surgical resection of diseased segments of lung usually did not change the over-all ventilation to any extent, as shown by external spirometry. Often, there were increases in the maximum breathing capacity after resection. Occasionally, there were decreases.

However, bronchspirometry showed that in the majority of cases (18 out of 24) there was some further loss of function on the diseased side after the surgical removal of the diseased segments of lung. These reductions in ventilatory function or in the per cent of the total oxygen absorbed were balanced by increases in these functions in the opposite undiseased lung.

In almost every case a reduction in the per cent of the oxygen absorbed on the operated side was associated with some postoperative complication such as empyema, atelectasis, or fever, attributable to no cause other than the chest lesion. Reductions in the per cent of the total air moved on the operated side occurred, on the other hand, following an apparently uneventful postoperative course. Nevertheless, it is possible for postoperative complications such as atelectasis and fever attributable to the chest lesion to occur and to have no change in the per cent of the oxygen absorbed or the per cent of the total air ventilated by the operated side.

REFERENCES

1. Cournand, A., and Richards, D. W.: Pulmonary Insufficiency. I. Discussion of a Physiological Classification and Presentation of Clinical Tests, *Am. Rev. Tuberc.* 44: 26, 1941.
2. Pinner, M., Leiner, G., and Zavod, W. A.: Bronchspirometry. III. The Functional Capacity of Normal Lungs, Severely Damaged Lungs, Lungs With Strictly Parenchymal Lesions, Thoracoplasty Lungs, and Re-expanded Pneumothorax Lungs, *J. THORACIC SURG.* 11: 241, 1942.
3. Baldwin, E. de F., Cournand, A., and Richards, D. W.: Pulmonary Insufficiency. I. Physiological Classifications, Clinical Methods of Analysis, Standard Values in Normal Subjects, *Medicine* 27: 243, 1948.
4. Norris, Charles M., Long, Joan, and Oppenheimer, Morton J.: Bronchspirometry: Apparatus and Technique, *J. THORACIC SURG.* 17: 357, 1948.
5. Robinson, W. L.: Bronchiectasis, A Study of the Pathology of Sixteen Surgical Lobectomies for Bronchiectasis, *Brit. J. Surg.* 21: 302, 1933.
6. Erb, I. H.: Pathology of Bronchiectasis, *Arch. Path.* 15: 357, 1933.
7. Kay, E. B., Meade, R. H., and Hughes, F. A.: Surgical Treatment of Bronchiectasis, *Ann. Int. Med.* 26: 1, 1947.
8. Lindskog, Gustaf E.: Changes in Pulmonary Volume Following Lobectomy for Bronchiectasis, *J. Clin. Investigation* 18: 251, 1939.
9. Birath, G., Crafoord, C., and Rudstrom, P.: Pulmonary Function After Pneumonectomy and Lobectomy, *J. THORACIC SURG.* 16: 492, 1947.

The Journal of Thoracic Surgery

VOL. 19

APRIL, 1950

No. 4

Original Communications

PNEUMONECTOMY FOLLOWED BY IMMEDIATE THORACOPLASTY

ROLAND K. IVERSON,* M.D., SAN FRANCISCO, CALIF. (BY INVITATION),
AND HOMER L. SKINNER,† M.D., STATEN ISLAND, N. Y.

WE RECENTLY reported three cases in which pneumonectomy and thoracoplasty had been performed at a single operation.¹ This series now includes ten patients. In this paper we should like briefly to review these cases, and to include also a few pertinent details of pre- and postoperative management as well as of technical considerations. First, however, it would seem appropriate to present those observations which prompted us to perform pneumonectomy and rather extensive thoracoplasty as a single procedure.

We all know how the encouraging early results of pulmonary resection in tuberculosis often gave way to extreme pessimism in the face of late spreads of disease in the contralateral lung following pneumonectomy, and in the ipsilateral portions of the remaining lung following lobectomy. Bronchial fistulas and tuberculous or mixed empyemas were not uncommon. Overdistention of the contralateral lung, resulting in reduced respiratory efficiency and reactivation of quiescent tuberculous foci, was particularly distressing in the pneumonectomy cases.

Improved surgical techniques and the advent of streptomycin have done much to eliminate the distressing complications of pulmonary resection in tuberculosis. In order to avoid the complications incident to overdistention of the contralateral lung, it has become routine practice in most thoracic clinics to follow pneumonectomy by one or two stages of thoracoplasty, and upper lobectomy by a single stage thoracoplasty. These operations are generally performed three to six weeks after the resection. Elective thoracoplasty, however, is not without risk. To quote Bailey and associates² in a recent article: "Since two of the five deaths were subsequent to elective thoracoplasties performed merely to obliterate the pleural space, it can well be imagined that we are on the lookout for a method of avoiding this secondary operation."

From the Surgical Service, United States Marine Hospital, Staten Island, N. Y.
Read at the Twenty-ninth Annual Meeting of The American Association for Thoracic Surgery, New Orleans, La., March 29-31, 1949.

*Surgeon, United States Public Health Service, San Francisco, Calif.

†Medical Director, United States Public Health Service, Staten Island, N. Y.

of a circle 2 inches in diameter. The instrument is made of malleable stainless steel, and is $\frac{3}{32}$ inch in thickness. The ligature slot opens at a distance of $\frac{1}{4}$ inch from the rounded distal tip and is cut obliquely.

TECHNIQUE

The ligature passer has been used in ligation of the pulmonary artery and veins in pneumonectomy and pulmonary lobectomy at the Montreal General Hospital.

After the appropriate segment of artery or vein has been dissected free, the tip of the unthreaded instrument is passed under the vessel. The ligature is then placed in the slot, and upon withdrawing the instrument, is carried about the vessel. It has been found most convenient to introduce the ligature into the slot in the following way: As the surgeon passes the tip of the instrument about the vessel to be ligated, an assistant holds the ligature under tension by grasping one end in a long curved forceps and the other end in his fingers. Upon placing the ligature in the slot the assistant releases the free end and the surgeon simultaneously withdraws the instrument, passing the free end of the ligature about the vessel (Fig. 2). Due to its malleability the surgeon can easily alter the curve of the instrument as occasion demands.

It is felt that this ligature passer may be used to advantage in a number of situations (for example, thorax, pelvis) where the structures to be ligated are deeply placed.

Acknowledgment.—Our gratitude is due Mr. Meredith Laflamme, of the Liquid Carbonic Corporation of Canada, Ltd., Montreal, for his painstaking craftsmanship in constructing several stainless steel models of the ligature passer.

Operative Technique.—Exposure for the combined operation is obtained through a long posterolateral incision, the proximal limb being carried somewhat higher than in the usual incision for a pneumonectomy alone. As recommended by Brock,⁸ we make the vertical limb of the incision close to the spinous processes. This permits both raising and closure of the trapezius and rhomboid muscles as a single layer. Blood loss also appears to be somewhat less with this incision.

The pleural cavity is entered through the periosteal bed of the sixth rib, following resection of a generous segment of this rib and the splitting of the fifth and seventh ribs paravertebrally. The intervening intercostal nerves and vessels are doubly ligated and divided. The posterior aspect of the hilus is then exposed as quickly as possible, the bronchus freed from the surrounding tissues, and a right angle clamp applied distally. Several 00 intestinal cotton sutures on an atraumatic needle are then placed high on the bronchus. These sutures are tied over the bronchial stump as the bronchus is being cut proximal to the clamp. No mattress sutures have been used in any case. This method of closure allows a high amputation and permits the stump to retract deeply into the mediastinum. In some of these cases it has been impossible to cover the bronchial stump with mediastinal pleura since complete parietal pleurectomy was performed. The pulmonary vessels are then treated in the usual manner, the artery being ligated and divided first, if possible, followed by the pulmonary veins. We have rarely used transfixion sutures distal to the proximal ligature in order to permit leaving as large a cuff as possible.

Following division of the hilar structures, the lung is freed from its remaining attachments to the chest wall and mediastinum. In so far as possible, care is taken to avoid breaking into thin-walled cavities and over these areas dissection was carried out in the endothoracic plane. This precaution particularly applied to large apical cavities. Bleeding from the raw chest wall has caused no problem since it is easily controlled by warm saline packs.

The phrenic nerve has been routinely interrupted, be it a right or left pneumonectomy.

The patient's condition is carefully evaluated at this point, and if considered satisfactory, thoracoplasty is immediately performed. In no patient have we failed to perform the thoracoplasty as planned, because of poor condition at the end of pneumonectomy. Even after such a formidable procedure as pneumonectomy, the addition of thoracoplasty did not produce shock or even an appreciable fall in blood pressure in any of these patients. It appears that performance of the additional operation at this time is not as shocking a procedure in itself as when performed over a closed and fairly rigid pleural space three to six weeks after pneumonectomy.

The usual thoracoplasty consisted of resection of the second through the seventh ribs. Generous posterior segments were removed but did not include the transverse processes. In some patients the second rib was left intact, but the intercostal structures were freed from the pleural surface and allowed to fall inward. It was our intention to preserve the normal chest and shoulder contours insofar as possible, and yet permit adequate obliteration of the pleural space.

Following thoracoplasty, the pleural cavity is thoroughly irrigated with saline and a No. 24 catheter inserted through a stab wound in the eighth intercostal space and posterior or midaxillary line. The intercostal structures, muscles, subcutaneous tissue, and skin were next approximated with interrupted cotton sutures and a lightweight but snug dressing was applied. One gram of streptomycin and 100,000 units of penicillin were instilled through the catheter, and the catheter clamped. In some of our recent cases no attempt has been made to reapproximate the intercostal structures. It is questionable whether this is a desirable procedure, and this will be elaborated upon later.

Immediate Postoperative Management.—Following completion of the operation, the patient is immediately placed on his back and the tracheobronchial tree thoroughly aspirated. In none of the cases was postoperative bronchoscopy necessary. We have abandoned the practice of routine postoperative bronchoscopy in this type of case as well as after other major thoracic operations. We feel that it unnecessarily prolongs anesthesia, and is no more effective than properly performed intratracheal aspiration with a catheter. We do not hesitate, however, to perform bronchoscopy on a patient should he remain "wet" after what is considered to be adequate intratracheal aspiration.

Minor³ in a recent article also stated: "When a thoracoplasty is performed over a closed pleural space containing air, or fluid, or both, a rapid increase in intrapleural pressure results. Highly positive pressure upon the heart and mediastinum produces a form of shock which may be rapidly fatal."

Our interest in pulmonary resection for tuberculosis is relatively recent, and fortunately we did not have this complication in any of our earlier cases. In a limited series, however, we did note the more benign course of postthoracoplasty resections as compared to those who had had no previous thoracoplasty. This occurred despite the increased technical difficulties which often presented themselves in a postthoracoplasty resection. There is also the problem of the patient who refuses postpneumectomy thoracoplasty, and who, by so doing, may definitely jeopardize his future well-being.

It is beyond the scope of the present paper to discuss the indications for pulmonary resection. Obviously the main indication for resection in pulmonary tuberculosis is thoracoplasty failure, and this group is necessarily absent from this series. Seven of the combined operations here reported were performed for extensive unilateral pulmonary tuberculosis or tuberculous empyema. One was performed for pulmonary cyst and lung abscess, one for squamous bronchiogenic carcinoma, and one patient had both squamous bronchiogenic carcinoma and active pulmonary tuberculosis (Table I).

TABLE I

CASE	PATIENT	AGE (YR.)	SIDE	INDICATION FOR RESECTION
1	N. P.	33	L	Far advanced pulmonary tuberculosis
2	R. K.	21	L	Far advanced pulmonary tuberculosis
3	F. A.	51	R	Pulmonary cyst, lung abscess
4	C. R.	62	L	Far advanced pulmonary tuberculosis (preoperative diagnosis: carcinoma)
5	A. G.	48	R	Bronchiogenic carcinoma
6	J. S.	38	L	Far advanced pulmonary tuberculosis
7	C. E.	62	L	Bronchiogenic carcinoma, pulmonary tuberculosis
8	J. C.	40	R	Far advanced pulmonary tuberculosis
9	L. H.	33	R	Far advanced pulmonary tuberculosis
10	A. G.	35	L	Tuberculous empyema, pulmonary tuberculosis

TECHNIQUE

Preoperative Management.—Tuberculous patients receive streptomycin for one to two weeks, and penicillin for twenty-four hours before surgery. The usual dosage of streptomycin was $\frac{3}{4}$ Gm daily. Premedication consisted of Nembutal gr. $1\frac{1}{2}$, one and one-half hours before operation, and morphine sulfate gr. $\frac{1}{4}$ and scopolamine gr. $\frac{1}{160}$ one hour before surgery.

Position on Table, and Anesthesia.—All of these patients were operated upon in the face-down position with the sound lung uppermost, as advocated by Overholt.⁴ Cyclopropane, oxygen, and ether were the anesthetic agents used. The patient was put in position on the face-down table as soon as the intratracheal tube had been placed and the course of anesthesia was proceeding smoothly. Intratracheal aspirations were carried out frequently.

The advantages of the face-down position have been enumerated by Overholt and others, but certain features deserve re-emphasis. The lessened danger of contralateral spread by gravity drainage is obvious. The ease and rapidity with which the bronchus can be exposed and occluded is also of extreme importance. It has also been our impression that patients operated upon in the face-down position show less tendency to develop shock or cardiac arrhythmias. To further combat the latter, intravenous procaine has been used prophylactically as a routine measure.

CASE 2.—R. K., white man, aged 21 years, was admitted May 12, 1948, from another facility where he had been hospitalized since May, 1947. Admission film revealed extensive infiltration and cavitation on the left, and an essentially clear right lung. Bronchoscopy in January, 1948, had shown moderately severe tuberculous involvement of the left-stem bronchus. Streptomycin therapy in dosage of 1 Gm. daily for six weeks produced marked subjective improvement but roentgen findings persisted.

Tomography revealed cavitation in both the left upper and lower lobes, one cavity being present anteriorly in the lingular segment of the upper lobe. Total thoracoplasty was considered but resection seemed preferable. The combined procedure was recommended. It should be noted that this patient observed the postoperative course of Case 1 closely, and then also requested the procedure himself, since he bitterly disliked the prospect of multiple operations.



Fig. 1 (Case 2).—Postoperative combined pneumonectomy and thoracoplasty. Note that both the first and second ribs are intact.

Streptomycin, $\frac{5}{8}$ Gm. daily, was started Aug. 19, 1948, and penicillin, 50,000 units every three hours, on Aug. 21, 1948. Left pneumonectomy and thoracoplasty were performed Sept. 1, 1948. In this patient, the first and second ribs were left in situ and the third through the seventh ribs were resected. The intercostal structures were freed from the medial aspect of the second rib and allowed to fall inward.

Postoperative management of this patient was exactly as that described and convalescence was completely uneventful. Temperature remained normal from the third postoperative day. Patient was up in a wheelchair on the second postoperative day and ambulant on the fourth postoperative day. In regard to ambulation, it must be mentioned that this patient had had a traumatic mid-thigh amputation of the left leg at the age of 6 years, and ambulation necessitated his wearing a rather cumbersome prosthesis. Cough and expectoration have been nonexistent since operation. Postoperative films showed a clear right lung and at no time any evidence of mediastinal displacement.

The anesthetic agent is discontinued during the skin closure, and a mixture of helium and oxygen administered. This mixture thoroughly removes the anesthetic agent from the lungs and facilitates the patient's early reaction after operation. The intratracheal tube is replaced by an oropharyngeal airway as soon as the patient starts to react. Nasal oxygen is started immediately. Blood is administered during the operative procedure in amounts up to 1,500 c.c. and continued after operation up to 2,000 to 2,500 c.c. A 500 c.c. transfusion was usually given on the first postoperative day. No unusual bleeding was encountered in any patient either during the operative procedure or in the postoperative period.

General Postoperative Considerations.—The same general principles apply to these patients as to others who have undergone major thoracic surgery. A few points will be emphasized.

Semi-Fowler's position and frequent turning were important to permit maximum pulmonary ventilation. Sedation in adequate amounts to control pain was necessary, and the patient was urged to cough and raise sputum at regular intervals. Coughing was greatly facilitated by having the patient sit up and apply pressure over the operated side. This could usually be started on the evening of the day of operation.

We particularly noted that these patients can cough more effectively in the early postoperative period than patients who have had pneumonectomy alone. This is attributed to the weight of the scapula and the pressure dressing over the decostalized area which render the mediastinum relatively stable.

Continuous suction of -10 cm. of water was maintained for forty-eight hours, except for a four-hour period on the first postoperative day following the instillation of 1 Gm. of streptomycin and 100,000 units of penicillin. These antibiotics were again given just before removal of the catheter on the second postoperative day.

Early ambulation is practiced and patients are urged to move about as much as possible after removal of the catheter and change of dressing on the second postoperative day. Sutures are removed on the sixth postoperative day and a pressure dressing is maintained for two to three weeks, or until the chest wall has become relatively firm.

The following case reports are presented in sufficient detail to illustrate the efficacy of the combined operation.

CASE REPORTS

CASE 1.—N. P., colored man, 33 years of age, was admitted April 16, 1948, following prolonged hospitalization at another facility. Chest film revealed extensive infiltration and multiple large cavities on the left, and infiltration in the right upper lobe. Tomography revealed a questionable small cavity in the right upper lobe, so the left pneumonectomy was deferred. Repeat tomography two months after admission failed to demonstrate any evidence of cavity in this region, and patient was prepared for surgery. Bronchoscopy revealed no evidence of significant endobronchial tuberculosis.

Streptomycin, 1 Gm. daily, was begun on July 5, 1948, and penicillin, 50,000 units every three hours, was started on July 13, 1948. After the usual preoperative preparation, left pneumonectomy and thoracoplasty were performed on July 14, 1948, in the manner described. Generous posterior segments of the second through the seventh ribs were resected. Convalescence was completely uneventful. Temperature remained normal from the ninth postoperative day and the highest temperature recorded in the postoperative period was 100.4° F. Bathroom privileges were allowed on the third postoperative day.

The wound healed per primam and excellent collapse of the left hemithorax was present. Postoperative films showed no mediastinal shift at any time and there was no evidence of contralateral spread.

The patient, in excellent general condition, was transferred back to the referring facility on Sept. 7, 1948. Sputum has remained negative by all methods of examination and pulmonary symptoms are absent. The patient has gained approximately fifteen pounds in weight and posture and arm function are excellent. He is now in a period of gradually increasing exercise preliminary to discharge.

This patient was discharged from the referring facility in January, 1949.

As noted in this case summary, it was decided to leave the second rib in situ in this patient because of his youth, and our desire to preserve normal arm and shoulder contours in so far as possible. This was accomplished, and at the same time adequate obliteration of the pleural space appeared to be accomplished. This patient has gained thirty-one pounds since operation, and when last seen for follow-up examination in March, 1949, the only noticeable deformity was the incisional scar.

CASE 3.—F. A., white man, 51 years of age, was admitted Aug. 3, 1948, with chief complaints of severe cough productive of 12 to 16 ounces of bloody sputum daily for three weeks, and weight loss of fifteen pounds.

There was a history of treatment in a Brooklyn hospital in 1942, for a right lower lobe abscess. He apparently completely recovered after five months' hospitalization, although chest films also revealed the presence of a large right upper lobe cyst.

His previous admission here was in October, 1946, when chest films revealed a huge right lower lobe abscess and a large right upper lobe cyst. Pneumonectomy was considered but seemed too hazardous in view of the patient's poor general condition, copious expectoration, and contralateral emphysema. Wide-open drainage was therefore instituted in November, 1946. Residual bronchopleural fistulas were closed by a pedicled muscle flap in March, 1947. Patient returned to full-time work as a crane operator in May, 1947, and remained well until three weeks before the present admission.

Chest film on the present admission revealed several small lower lobe abscess cavities and the right upper lobe cyst which showed no apparent change from previous films. The right lung was contracted and the left lung moderately emphysematous.

Patient was placed on penicillin by both intramuscular and aerosol routes with marked improvement in condition, particularly in reduction of sputum output. He was prepared for surgery and on Sept. 8, 1948, right pneumonectomy and thoracoplasty were performed. The lung was completely "frozen" to the chest wall and dissection was long and tedious. Despite this, the patient tolerated pneumonectomy extremely well, and because of the barrel type of chest, resection of long posterior segments of the second through the eighth ribs was necessary to produce satisfactory collapse.

Convalescence was uneventful. Temperature reached a high of 101.4° F. on the second postoperative day and returned to normal on the fourth day. Cough and expectoration were negligible after operation. When he was last seen for check-up examination, general condition was excellent and the only complaint was moderate dyspnea on exertion.

It would appear that in this type of case with contralateral emphysema, immediate thoracoplasty would prevent any further overdistention of the already hyperinflated lung, and thereby insure the patient's continued well-being. This case also illustrates the fact that even after a prolonged tedious pneumonectomy, the addition of thoracoplasty appears to be well tolerated.

CASE 4.—C. R., colored man, aged 62 years, was admitted on Sept. 21, 1948, following short periods of hospitalization at three different institutions since July 21, 1948. Chief complaints on admission to first hospital were pain in left chest, cough, epigastric distress, and weight loss of thirty pounds. Chest films revealed a large homogeneous density in the left mid-lung field. Sputa were negative for acid-fast bacilli. He was transferred to a second hospital where exploratory thoracotomy was performed through an anterior approach on Aug. 12, 1948. The impression was inoperable bronchiogenic carcinoma with superimposed pulmonary tuberculosis, the latter diagnosed by biopsy of a peripheral nodule. No tumor biopsy was obtained.

On admission to this hospital the findings were much as those previously noted. It was felt, however, that patient should be re-explored in view of bronchoscopic and x-ray evidence of probable operability and failure to demonstrate any metastases. Sputa examinations also continued to be negative for acid-fast bacilli. The preoperative diagnosis was bronchiogenic carcinoma.



Fig. 2.

Fig. 2 (Case 2).—Postoperative appearance three weeks after combined pneumonectomy and thoracoplasty.

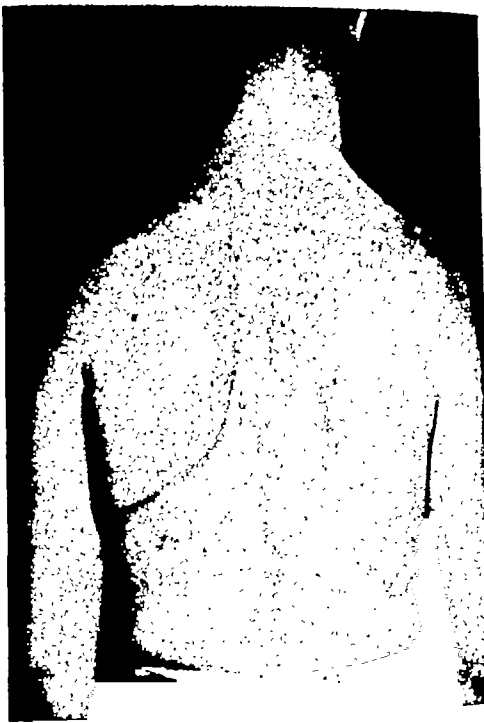


Fig. 3.

Fig. 3 (Case 2).—Same patient six months after operation.



Fig. 4.

Fig. 4 (Case 3).—Preoperative film showing large right upper lobe cyst, right lower lobe abscess, and emphysema in the contralateral lung.



Fig. 5.

Fig. 5 (Case 3).—Postoperative view of patient showing minimal deformity despite resection of the second through the eighth ribs. Previous scars from abscess drainage and myoplastic closure of bronchocutaneous fistula in 1946.

On Oct. 13, 1948, a right pneumonectomy and right thoracoplasty were performed with resection of the third through the seventh ribs, and stripping of the second rib.

Immediate postoperative course in this patient was extremely satisfactory. He continued to do well for about four weeks, after which he began to run a low-grade fever. Thoracentesis yielded purulent pleural fluid. This failed to clear under antibiotic therapy and aspiration, and on Dec. 2, 1948, a tube was inserted into the pleural cavity. At no time was a bronchial fistula demonstrated. On Dec. 30, 1948, an anterolateral thoracoplasty was performed and a tube again left in place in the pleural cavity. This tube was gradually removed in the ensuing two weeks and the empyema cavity was completely obliterated. General condition continued to improve and patient was discharged on Feb. 7, 1949.

It is difficult to explain why this patient developed an empyema four weeks after the operation, except that he may have had an extremely small bronchial fistula which promptly closed spontaneously. Such a fistula, however, could never be determined even by instillation of dye. It is interesting to note that even though this empyema developed postoperatively, it was very easy to treat. In view of the fact that major collapse of the right hemithorax had already been accomplished, it was necessary only to do a small anterolateral thoracoplasty and institute tube drainage. It was plainly evident in this patient that we had not removed as long segments of rib, particularly as regards the third and fourth ribs, as on the earlier patients.

CASE 6.—J. S., colored man, aged 38 years, was admitted Nov. 4, 1948, for surgical therapy of extensive pulmonary tuberculosis of the left lung. Onset of disease was in March, 1948. From July 11 to Aug. 10, 1948, patient received 64 Gm. of streptomycin with little improvement. Admission x-ray examination at this hospital revealed extensive cavitation, thickened pleura, and possibly pleural effusion on the left. Tomography revealed no evidence of cavitation on the right. Bronchoscopy revealed no significant endobronchial disease.

After the usual preparation, left pneumonectomy and thoracoplasty were performed on Nov. 30, 1948. The lung was found to be boggy and firm in consistency throughout, and completely "frozen" to the chest wall and pericardium. Pneumonectomy necessitated complete parietal pleurectomy and partial pericardiectomy. Despite a prolonged tedious operation, thoracoplasty was immediately performed, the patient tolerating both procedures extremely well. Despite gross contamination of the pleural cavity at the time of operation (the lung literally exuded pus and there were pockets of pus between the lung and pericardium) there was no postoperative wound infection, or infection in the pleural cavity. The right lung remained clear, and sputa have been negative for tubercle bacilli by all methods since operation. His present general condition is excellent. He is gaining weight and posture and arm function are good.

This pneumonectomy was probably the most difficult technically of all the cases in this series. Removal of the lung without contamination of the pleural cavity was impossible. Here, again, despite a prolonged difficult operation, the patient tolerated the addition of thoracoplasty extremely well. It is felt that the addition of the thoracoplasty greatly enhanced his smooth postoperative course, and lessened the likelihood of wound complications.

CASE 7.—O. F., white man, aged 62 years, was admitted Nov. 16, 1948, with chief complaints of hemoptyses, cough, and weight loss. Onset of present illness dated back two years, when patient first noted cough and hemoptyses. He continued to feel well except for the hemoptyses. Chest roentgenograms were negative, and careful examination at that time for acid-fast bacilli was negative. He remained fairly well until about four months prior to admission when he first noted extremely poor appetite and beginning weight loss. Dyspnea also appeared which had not been present before.

Three weeks prior to admission bronchoscopy was done at a private hospital in Manhattan and no significant findings were noted. His physician then advised hospitalization for further observation.

Thoracotomy was performed through a posterolateral approach on Oct. 7, 1948. A large firm mass involving both upper and lower lobes posteriorly was found and this was densely adherent to the chest wall and aorta. The hilar structures, however, were free of nodes and tumor involvement, so left pneumonectomy was performed followed by immediate thoracoplasty as described. Again the second rib was left intact.

Convalescence was completely uneventful. Temperature remained at normal levels after the fifth postoperative day and the patient was ambulant after the second postoperative day.

The pathologic report revealed extensive caseous pulmonary tuberculosis involving about one-half of the pulmonary parenchyma. No carcinoma was found.



Fig. 6.

Fig. 6 (Case 4).—Extensive lesion left midlung field. Had had exploratory thoracotomy at another institution with diagnosis of inoperable carcinoma and pulmonary tuberculosis.



Fig. 7.

Fig. 7 (Case 4).—Postoperative combined pneumonectomy and thoracoplasty. Note excellent collapse despite presence of the first and second ribs, the latter was stripped on its pleural surface.

This patient was returned to the referring hospital shortly after operation, and at the present time is on a program of graduated exercises preliminary to discharge. He has continued to remain well and has gained in weight and strength. Posture and arm function are excellent.

CASE 5.—A. G., white man, aged 48 years, was admitted to this hospital Sept. 18, 1948, complaining of cough and chest pain for approximately two weeks, and chills and fever for about four days prior to admission. There was a history of moderate weight loss, and also history of several episodes of hemoptyses within the few days prior to admission.

Patient was acutely ill on admission with considerable dyspnea and signs of consolidation in the right lung. Bronchoscopy on Oct. 2, 1948, revealed a tumor mass protruding from the dorsal segment of the right lower lobe and completely blocking the right middle lobe bronchus also. Biopsy revealed squamous-cell carcinoma.

sputa have remained negative for acid-fast bacilli. He is now on a program of increasing activity at the sanatorium where he was transferred following operation. Prolonged convalescence because of the tuberculosis is not indicated, since the pathologic report revealed the hilar nodes to be involved with tumor, making his ultimate prognosis poor.

It would appear that we were extremely radical in performing an extensive thoracoplasty on a patient with carcinoma who was not in the best physical condition. However, the bronchial closure in this patient did not appear to be satisfactory, and could not be made so because it was amputated extremely high and the tissues appeared to be friable from chronic inflammation. Even in this elderly patient, posture and arm function following this procedure are excellent.



Fig. 10 (Case 7).—Postoperative view of patient showing excellent arm and shoulder function

CASE 8.—J. C., colored man, aged 40 years, was admitted Jan. 18, 1949. There was a history of being well until April, 1948, at which time he experienced generalized malaise, weight loss, and developed a productive cough, later associated with hemoptyses. He was hospitalized at another facility where x-ray examination revealed extensive tuberculosis involving the right lung. Right pneumothorax was attempted without success.

Chest film and tomography on admission revealed multiple cavities in the right upper lobe and extensive consolidation and questionable cavitation in the lower lobe. Bronchoscopy revealed endobronchial disease involving the right upper lobe bronchus.

Following preliminary streptomycin therapy, right pneumonectomy and right thoracoplasty were performed on Feb. 3, 1949. Complete extrapleural dissection was necessary over the entire upper half of the lung. Following pneumonectomy, a thoracoplasty was performed, with resection of the third through the seventh ribs; the second rib was stripped medially, and the intercostal structures allowed to fall inward. Here again no attempt was made to approximate the intercostal structures. Operation was completed in the usual fashion and patient withstood both procedures extremely well.

This man's convalescence has been considerably more stormy than the previous patients, with chief complaint being dyspnea. No significant shift of the mediastinum has been observed on any films and the left lung has shown no evidence of spread. How-

Physical examination on admission revealed a chronically ill man, with evidence of considerable weight loss. There was evidence of consolidation over the left upper lobe, and this was confirmed by x-ray examination. Bronchoscopy on Nov. 18, 1948, revealed marked retraction of the left upper lobe bronchus but no tumor was seen. Bronchoscopy was repeated on Dec. 6, 1948, at which time examination with a right-angle lens revealed a tumor mass within the left upper lobe bronchus. Biopsy was obtained which revealed squamous carcinoma, grade 2. It should be noted that sputum examinations were consistently positive for acid-fast bacilli after admission to this hospital, but that the x-ray picture and clinical course were so characteristic of carcinoma that exploration was contemplated despite the persistent presence of acid-fast bacilli.

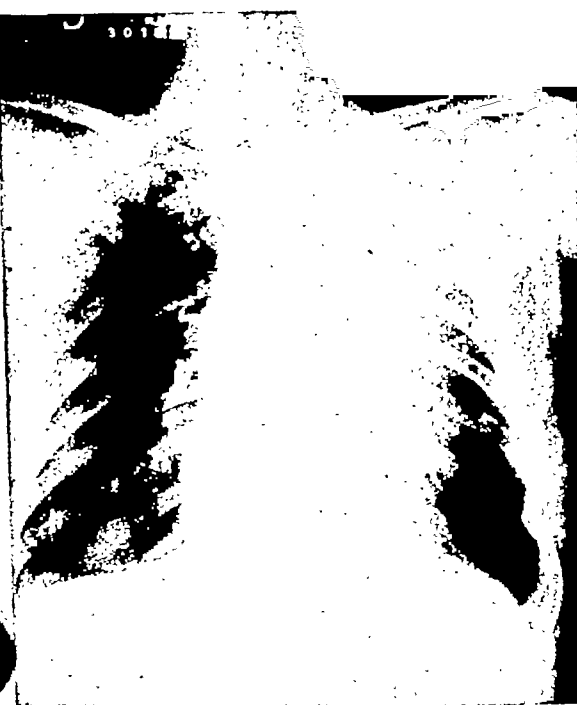


Fig. 8.

Fig. 8 (Case 7).—Preoperative film showing extensive left upper lobe lesion. Sputum positive for acid-fast bacilli. Positive bronchoscopic biopsy squamous carcinoma left upper lobe bronchus.



Fig. 9.

Fig. 9 (Case 7).—Postoperative combined pneumonectomy and thoracoplasty. Small amount of fluid still present in residual pleural space.

After the usual preoperative preparation, thoracotomy was performed on Dec. 21, 1948. The tumor mass encroached upon the hilar region, particularly in the superior pulmonary vein, but intrapericardial ligation of this vessel greatly facilitated removal. Several nodes were removed from the mediastinum and subaortic region, which appeared to be involved in carcinoma. The wall of the bronchus also appeared to be involved, almost up to the carina, and an extremely high amputation was necessary. Bronchial closure was extremely difficult and was not as satisfactory as desired. For this reason it was decided to perform a thoracoplasty, immediately, in the hope of lessening any probable postoperative pleural complications. To secure adequate collapse, generous posterior segments of the third through the eighth ribs were resected and the second rib was stripped on its medial surface. The intercostal structures were then allowed to fall directly over the mediastinum and no attempt was made to approximate the upper and lower flaps.

Postoperatively this patient did extremely well. He was ambulant after removal of the tube on the second postoperative day, and the highest postoperative temperature was 99° F. His present condition is excellent, he is gaining in weight and strength, and

the same type of material which was aspirated from the pleural cavity. General condition became progressively worse, appetite decreased, weight dropped approximately sixteen pounds, and he noted daily temperature elevations.

On admission to this hospital x-ray examination revealed an extreme collapse of the left lung and the pleural cavity filled with fluid. Aspiration yielded grossly purulent fluid which was highly positive for acid-fast bacilli. Sputa examinations were persistently positive for tubercle bacilli.

Tomography failed to reveal evidence of cavitation in the right lung and after preliminary streptomycin therapy, left pneumonectomy and left thoracoplasty were performed on March 14, 1949. Convalescence to date has been extremely satisfactory, although it is as yet too early to make any final predictions.

SUMMARY

Ten patients who have undergone total pneumonectomy and rather extensive thoracoplasty at a single operation, have been presented, together with a brief description of the operative procedure and a few pertinent details of pre- and postoperative management. It would appear that this combined procedure has its chief advantage in the patients with pulmonary tuberculosis requiring pneumonectomy, in whom overdistention of the contralateral lung is particularly undesirable. We have extended indications for the procedure to include other patients in whom it would appear desirable to follow pneumonectomy with a thoracoplasty, in particular those with contralateral emphysema or those patients in whom postoperative pleural complications might be expected.

Our experience seems to indicate that the thoracoplasty may safely be performed upon completion of the pneumonectomy, if proper attention is given to supportive therapy and certain operative details. The necessity for only one operation and the lessened hospital stay are particularly desirable.

Although we did have one bronchial fistula occurring in this series, it would appear, theoretically at least, that prompt obliteration of the pleural space should greatly lessen the dangers of bronchial fistula and empyema, both as to their incidence and their treatment, should they occur.

Our observations of these cases also seem to indicate that disturbances in respiratory physiology are less marked than after pneumonectomy which is not followed by thoracoplasty. Decostalization of the chest wall of sufficient degree to permit the scapula to fall inward and the supplementing of this with a pressure dressing appears to render the mediastinum relatively stable. By the same token the coughing mechanism of the patients in the immediate postoperative period appears to be enhanced.

The posture and arm function of these patients appears to be surprisingly good. In patients who have gained weight following the procedure, little deformity is present.

There has been no mortality in any of the patients upon whom this combined procedure has been used.

REFERENCES

1. Iverson, R. K., and Skinner, H. L.: Pneumonectomy Followed by Immediate Thoracoplasty—Preliminary Report, Pub. Health Rep. 64: 140, 1949.
2. Bailey, C. P., Tropea, E., Jr., and Rubenstein, L. H.: Recent Advances in Pulmonary Surgery, S. Clin. North America 27: 1373, 1947.

ever, he has continued to require the occasional use of nasal oxygen, particularly at night. He has been afebrile since shortly after operation, and aspirated specimens from the right pleural cavity have failed to reveal any evidence of growth, either acid-fast bacilli or secondary organisms. His general condition has remained good and it is felt that this patient will eventually make a good recovery despite the dyspnea now present. It is probable that bronchspirometric determinations prior to operation in this patient would have revealed considerably more loss of function in the so-called good lung, than was estimated prior to surgery.

CASE 9.—L. H., white man, aged 33 years, was admitted Jan. 13, 1949. There was a history of pulmonary symptoms since January, 1947, but patient did not seek medical attention until the spring of 1948, when progressive weakness, hemoptyses, and marked weight loss occurred. He was hospitalized at another facility where x-ray examinations revealed bilateral infiltrations with multiple cavities in the right upper lobe and probably in the apex of the right lower lobe. Right pneumothorax was initiated in May, 1948, followed by right pneumonolysis in June, 1948. Despite a complete pneumonolysis and excellent collapse of the right lung, cavities failed to close. In addition, the patient was extremely uncooperative and left the hospital against advice, after a few months. He was admitted to another hospital in August, 1948, where pneumothorax was continued, and despite a fairly stable clinical course, patient failed to convert sputum, and cavities were still evident.

Examination on admission revealed a chronically ill white man, in no obvious distress. Rather extreme collapse of the right lung was present, but cavities were evident even on plain films and tomography revealed multiple cavitations in the collapsed lung. Sputum was positive for acid-fast bacilli. Bronchoscopy on Jan. 19, 1949, revealed no evidence of active tuberculous ulceration but there was some narrowing noted in the orifice to the right middle lobe, which was interpreted to be due to a healed tuberculous process. The right pneumothorax showed no tendency to re-expand.

On Feb. 16, 1949, right pneumonectomy and thoracoplasty were performed in the manner described, with removal of the second through the seventh ribs. The intercostal structures were loosely sutured against the posterior chest wall, but were not approximated at the site of the rib resection.

Postoperative management was similar to cases previously described. The immediate course was extremely satisfactory, but two weeks after operation the patient developed signs of a bronchial fistula and coughed up clear serous material from the right pleural cavity. On March 3, 1949, through an anterolateral incision, anterior segments of the second through the fifth ribs were resected, and the pleural cavity opened through the periosteal bed of the third rib. The residual pleural cavity was found to be small, was covered with clean granulations, and contained a small amount of clotted fibrin and a small amount of amber fluid. Despite the previously noted clinical presence of a bronchial fistula, there was no visible evidence of one even on careful examination. Resuture of the bronchus seemed impossible since it had retracted so deeply into the mediastinum. A No. 24 catheter was inserted and brought out through a stab wound anteriorly. The wound was closed in the usual fashion and a pressure dressing applied. Convalescence from this procedure has been extremely satisfactory, and cough and expectoration have progressively decreased until at the present the amount of sputum raised is minimal. Sputum has continued to be negative for acid-fast bacilli since operation, and it is anticipated that the catheter can be removed completely in the next two to three weeks.

CASE 10.—A. G., white man, aged 35 years, was admitted Jan. 10, 1949. There was a history of tuberculosis since January, 1938. In 1939, he had a small upper-stage thoracoplasty, and for reasons known only to the doctor under whose care he was at that time, pneumothorax was instituted following the first-stage of thoracoplasty, and further operations abandoned. Pneumothorax was apparently successful and patient returned to work in 1940, continuing the pneumothorax on an outpatient status. He felt well until November, 1948, when he developed weakness, exertional dyspnea, and cough. The pleural cavity was aspirated by a private physician, and the patient later noticed that he coughed up

FUNNEL CHEST AND ALLIED DEFORMITIES OF THE THORACIC CAGE

CHARLES W. LESTER, M.D.
NEW YORK, N. Y.

FUNNEL chest is not a particularly uncommon deformity and a definite number of those displaying it are severely incapacitated by it. There is a considerable literature on the subject dating back to the sixteenth century, admirably reviewed by Ochsner and DeBakey¹ in reporting a case. The etiology was obscure and the surgical treatment unsatisfactory, however, until Brown² recognized a short central tendon of the diaphragm as the cause of the deformity and described a surgical method for its correction. During the past five years we have seen a great many of these cases and have operated upon 31 of the patients.

The deformity is produced by a short central tendon of the diaphragm which flattens the dome of the relaxed muscle so that when the muscle contracts during inspiration the lower end of the sternum, to which the diaphragm attaches, is drawn backward causing, in time, a funnel-shaped deformity of the chest wall whose apex is at the xiphoid. The deformity may be symmetrical or asymmetrical and its character is determined by the portion of the central tendon which is shortest (Figs. 1 and 2). It may be considered under three heads: first, that produced by the direct pull of the diaphragm on the xiphoid and costal arch; second, that which appears elsewhere on the thoracic cage as a result of this pull; and third, the abnormalities of the organs within the chest as a result of the deformity of the thoracic cage.

When the mid-portion of the central tendon is shortest the xiphoid and lower end of the sternum will be retracted so that the sternum curves backward from the second segment to the xiphoid, where the depression is greatest. The costal cartilages are also drawn backward by the retraction of the sternum. This applies not only to the single cartilages but also to the fused cartilages forming the costal arch. The result will be a symmetrical funnel (Fig. 3).

If the diaphragm is short in the lateral leaflets as well as in the mid-portion of the tendon there will be a direct pull on the costal arch as well as on the xiphoid, and in addition to the funnel there will be a transverse groove across the anterior chest wall below the xiphoid. This resembles the Harrison's groove of rickets, although rickets has nothing whatever to do with funnel chest (Fig. 4).

If one lateral leaf of the diaphragm is shorter than the other the deformity will be asymmetrical with the maximum depression on the side of the greater shortening. This will cant the sternum toward that side as well as retract its lower end (Fig. 5).

If the shortening is in the lateral leaflets and not in the mid-portion of the central tendon there will be a groove without a funnel and it will be unilateral or bilateral depending on whether one side or both are short (Fig. 6).

Read at the Twenty-ninth Annual Meeting of The American Association for Thoracic Surgery, New Orleans, La., March 29-31, 1949.

out several weeks following pneumonectomy. We believe now that thoracoplasty not only should be done early but that it should be more complete than the operation we have carried out previously. We feel that the first rib should be removed and that, although it probably is not necessary to remove the transverse vertebral processes, the procedure should be carried down low enough to accomplish complete obliteration of the pleural cavity on that side.

DR. CARL SEMB, Oslo.—Taking advantage of being now a member of the Association, I should like to mention something about the combination of resection and thoracoplasty. From a theoretic point of view, as has been pointed out by others today, it is of great importance to perform a thoracoplasty to reduce the thoracic cage according to the resected part of the lung; the remaining part of the lung should not be distended. Thus in a prolonged resection and a prolonged thoracoplasty on the lower lobe we may do it with a phrenic nerve avulsion or with total resection and total thoracoplasty.

I have usually started with thoracoplasty in cases of tuberculosis because of the efficiency of the thoracoplasty under different conditions; especially where there are indications for lung resection with bronchiectasis we have, as Dr. Sanger pointed out, a very good effect with thoracoplasty if there is a total bronchiectasis. If we start with thoracoplasty we have the chance of being able to cure, or nearly cure, the patient without doing resection of the lobe or the lung afterward. When we have started with thoracoplasty, this does not usually cause difficulties so far as later resection of the lung is concerned, and it is not much more difficult than usual. The main indication for thoracoplasty is, I think, in such cases to start with thoracoplasty and afterward do a resection.

DR. SKINNER (closing).—I wish to thank the members for discussion of this presentation. I would like to emphasize two points, first, this procedure is not quite so formidable as it may at first appear. In none of these cases have we incurred any degree of shock following the thoracoplasty procedure. Of course in all these patients a careful review has to be taken with the anesthetist to evaluate their condition before proceeding with the thoracoplasty. I might say that three of the patients were not particularly good risks; they had massive empyema and complete extrapleural dissection was necessary, which was quite long and tedious; in one the operation lasted more than five hours. But as a rule thoracoplasty does not take more than 20 to 30 minutes. The other point I would like to make is concerning the care of complications. One patient had a bronchopleural fistula and one had an empyema pocket. The ease with which these were handled and the very little discomfort to the patient, if compared to a case without thoracoplasty, is a remarkable thing; either one could have been of major consequence. So we feel that is one advantage of the procedure.

The second class of deformity is that resulting from the alteration of the ribs and vertebral column because of the funnel sternum. The pull of the diaphragm seems to exaggerate the normal curve of the dorsal spine giving the individual a round-shouldered appearance and also making the funnel seem deeper. In addition the downward slant of the ribs is increased, thus making a long and narrow chest with diminution of both the anteroposterior and lateral diameters (Fig. 7). This gives a characteristic appearance to the roentgenogram and is an important consideration in predicting the result of corrective surgery.

The structural changes in the bony thorax can be expected to produce some effect on the thoracic organs. The lung is somewhat compressed but this is a minor disturbance and seldom amounts to more than 25 per cent reduction of the lung volume. It may also be distorted and it is possible that the respiratory infections to which these patients are unduly susceptible may be explained on the basis of defective bronchial drainage due to this distortion. The breathing is paradoxical to the extent that the lower end of the sternum and costal arch are retracted backward during inspiration and move outward during exhalation.

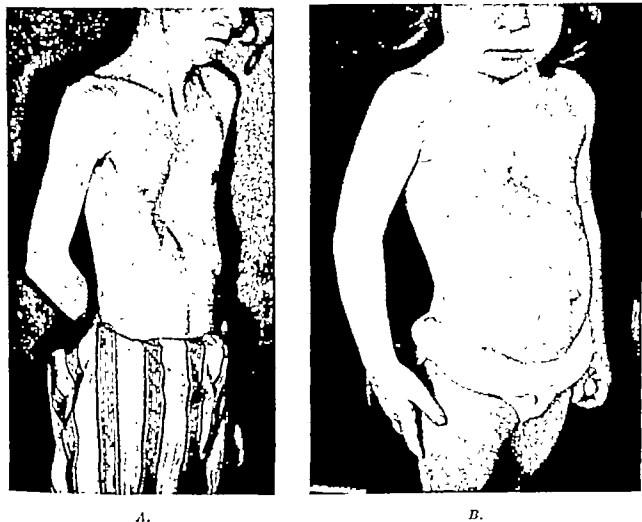


Fig. 3.—A, Funnel chest in boy 19 years of age. B, Funnel chest in girl 3 years of age.

The heart is the organ most effected by the deformity. It is displaced to the left, often to such an extent that the apex is in the axilla, and is also compressed. This compression interferes with the dilation of the heart necessary to accommodate the increased blood flow associated with muscular activity and hence may have a profound effect on the patient's exercise tolerance. Dyspnea associated with funnel chest is due to cardiac restriction and not pulmonary

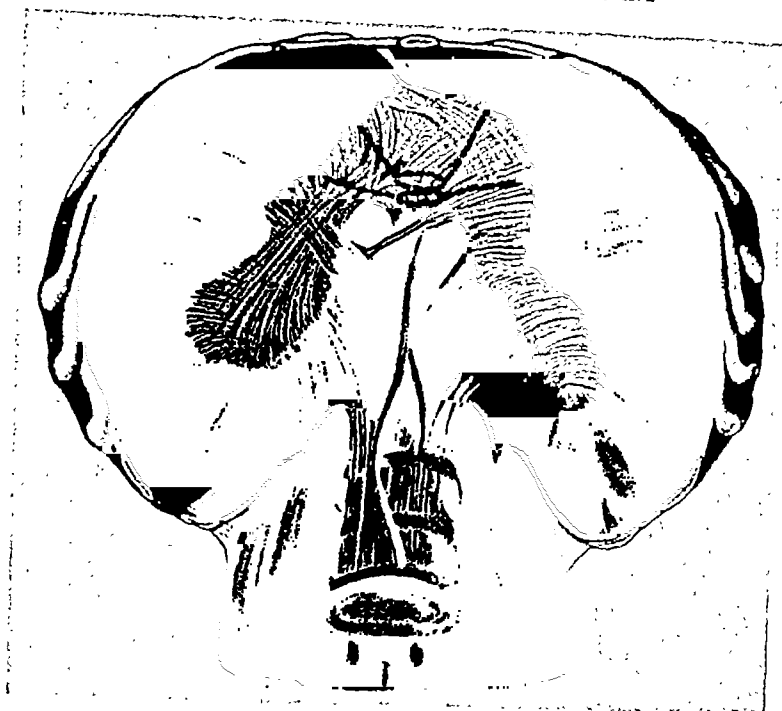


Fig. 1.—Diagrammatic sketch of diaphragm in normal position. First broken line indicates position of sternum with shortening of mid-portion of tendon only. Second broken line indicates position when lateral leaflets are also shortened.

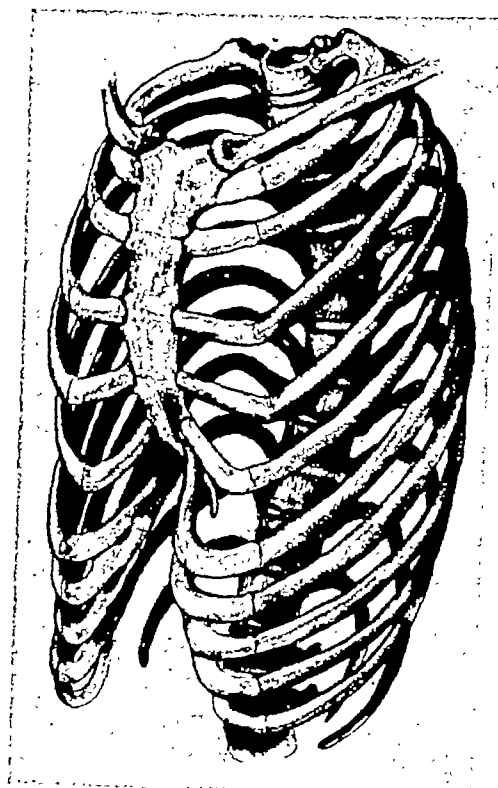


Fig. 2.—Diagrammatic sketch of bony thorax showing deformity produced by shortening of central tendon of diaphragm.

compression. A systolic murmur is also common and many of these patients are treated for cardiac disease which does not exist. Electrocardiographic changes are usual but their significance is not understood and they are now the subject of investigation.

Obviously a deformity of this sort will alter the appearance of the breasts in the postadolescent female. If the deformity is symmetrical the breasts may seem larger because of the deepening of the groove between them. If the deformity is asymmetrical one breast will be in a depression and hence will appear smaller (Fig. 5). In two cases of this sort the depressed breast was actually smaller and its elevation to the level of the other breast did not entirely satisfy the owner although it did materially decrease the deformity.



Fig. 6.



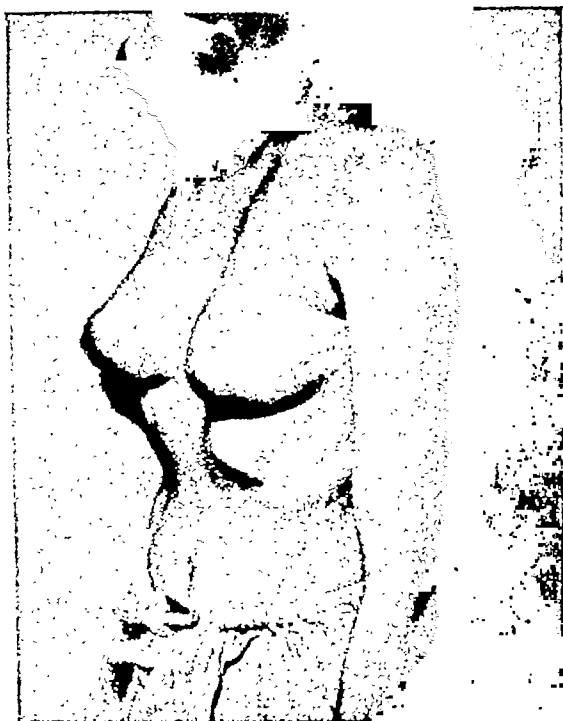
Fig. 7.

Fig. 6.—Transverse groove without funnel chest depression in boy 4 years of age.

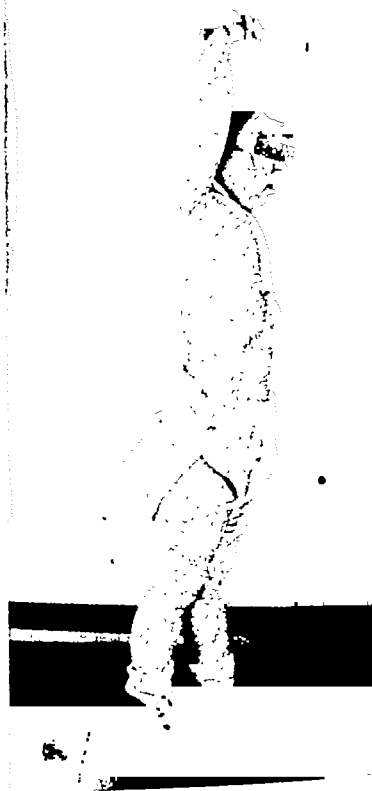
Fig. 7.—Roentgenogram of chest showing abnormal downward slant of ribs, narrowing of thorax, increased bronchial markings, and displacement of heart.

Nearly all the patients with obvious funnel chest are also underweight. This, together with the round-shouldered, narrow-chested, and funnel-chested appearance of the individual, gives him anything but a robust figure. The deformity is least at birth and increases with the growth of the child. It first affects the lower sternum and adjacent cartilages but sooner or later involves the ribs and vertebrae to produce distortion of the bony cage. Therefore, the time to correct the deformity is before it involves bony structures other than the sternum and thus becomes irreversible.

The indications for operation are physiological and psychological. In the extreme case with greatly limited activity and defective physical development there can be no question as to the advisability of operation. When the exercise

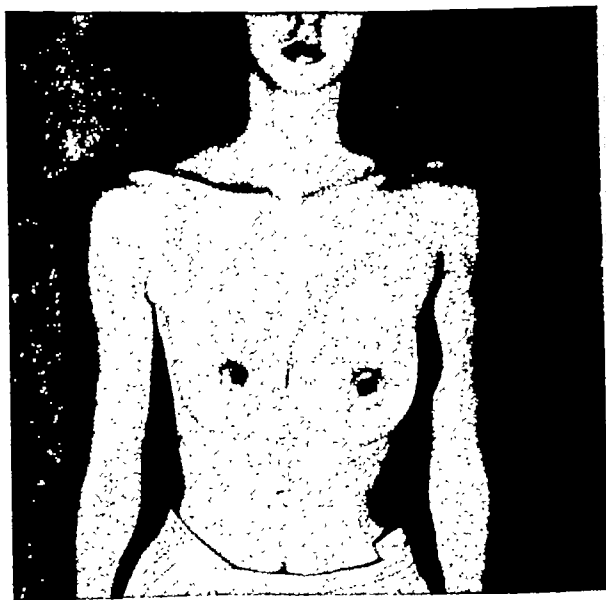


A.



B.

Fig. 4.—A, Well-developed transverse groove in funnel chest deformity in girl 15 years of age. B, Early transverse groove in funnel chest in girl 2 years of age.



A.



B.

Fig. 5, A and B.—Asymmetrical deformity of funnel chest producing apparent underdevelopment of right breast, corrected by operation.

retracted laterally. The pectoralis major muscles are separated from their sternal attachments and likewise retracted. The rectus abdominis muscles are split longitudinally through sheath and muscle to expose the costal arch (Fig. 8). Because the costal arch is narrow and the sides drawn towards the midline by the deformity an adequate exposure can be obtained without undue retraction. The cartilages are resected subperichondrially and this

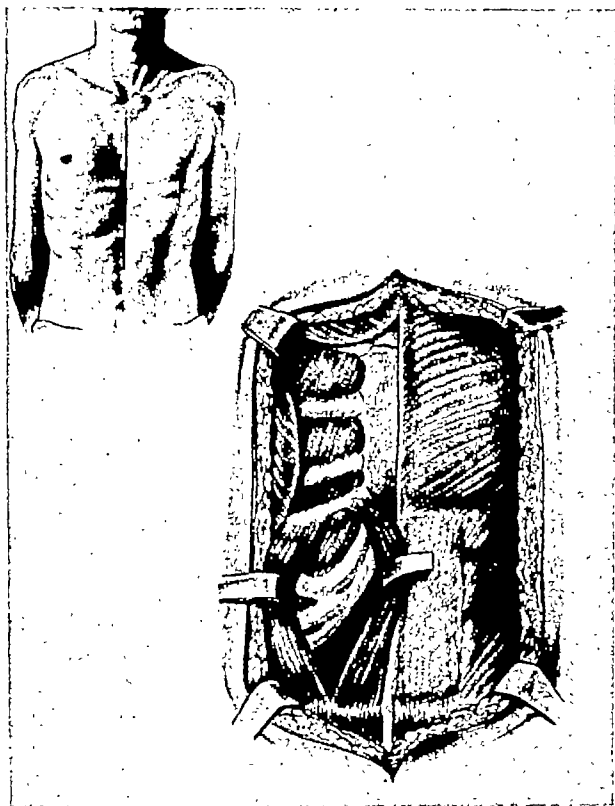


Fig. 8.—Radical operation. Exposure for resection of cartilages. In the right half the pectoralis major has been separated from the sternum and retracted exposing the individual costal cartilages below the second. The rectus abdominis has been split and retracted to expose the costal arch, the narrow arch making this exposure possible. Incision shown in insert.

is a slow and tedious procedure which must be done with great care to avoid tearing the pleura. The perichondrium is left so that the cartilages will regenerate. The wedge osteotomy is done with a small hand skull saw and a chisel. Stainless steel wires, 22 gauge, are used instead of silver because they are stronger. In a large boy or an adult one or both wires should be double.

tolerance is only moderately decreased, operation is also indicated. If the deformity is first noted in infancy and does not decrease with age, or becomes more pronounced, surgical interference is still advisable to prevent an irreversible deformity. Furthermore, although there is no apparent decrease in the exercise tolerance before operation, because the patient has never known anything different, he will realize after operation that a limitation did exist because he can then exercise more. The patient with moderate deformity without functional disturbance will not be benefited by operation except for psychological reasons.

The psychological aspect of the situation is of great significance. The girls do not pay much attention to the deformity until their breasts begin to develop and they start to wear a brassiere. Then they become conscious of it. The boys notice their deformity earlier probably because they run around unclad with other boys who take delight in calling attention to any deformity with derisive language. Many children take this teasing without trouble but others cannot and they are entitled to a corrective operation.

The operative procedures have been described by Brown² and we³ have reported on our modification of them. A simple operation to be used on infants consists in separating the xiphoid from the sternum and allowing the diaphragm to pull it back. This is done through a longitudinal incision over the lower end of the sternum and adjacent abdomen. The xiphoid is hidden by the cartilages of the costal arch which are drawn together by the deformity and it is necessary to resect the apex of the arch to expose it. The xiphoid is then detached from the sternum by a transverse incision which enters the ventral mediastinum and thus severs the substernal ligament, a prolongation of the linea alba along the under surface of the sternum. The diaphragmatic attachments to the arch in the immediate vicinity are then pushed off and the wound closed.

The radical operation must be used in all instances where the deformity is established and that will probably be in all patients more than 16 months old. The procedure we now use has been developed by a process of trial and error. Since the costal cartilages below the second are responsible for the deformity as well as the sternum, they must be resected laterally to the point at which the backward curve commences. The cartilages of the costal arch must also be resected to the point at which the backward curve commences and far enough to take in the lower border of the transverse groove, if this exists, that is, to include the third to the eighth or ninth cartilages—something we did not realize at first (Fig. 8). The xiphoid is then detached from the sternum, the ventral mediastinum entered, and the loose tissue pushed off the posterior surface of the sternum. No attempt is made to separate the substernal ligament from the sternum as its division, when the ventral mediastinum is entered, is sufficient. A transverse wedge osteotomy is then done across the third sternal segment through the anterior table only, leaving the posterior table intact, and the sternum is elevated to a normal position by producing an incomplete fracture along this line. Two pairs of holes are drilled in the lower end of the sternum and two stainless steel wires threaded through these holes across the back of the sternum (Fig. 9). The ends of the wires are brought through the skin and muscle on each side of the incision and the wound closed. The elevated position of the sternum is maintained by traction on the wires whose ends are then fastened into an external appliance placed transversely across the chest.

Some of the technical details of the operation need further elucidation. A longitudinal midline incision is made from the level of the second sternal segment to a point about 4 cm. above the umbilicus. The skin and subcutaneous tissues are dissected up and

The ladder is left on for two weeks. By this time the chest wall has stiffened sufficiently so that paradoxical breathing will not take place except during forced respiration. If the patient has a cough or is a screaming child it will be safer to leave the ladder on a few days longer.

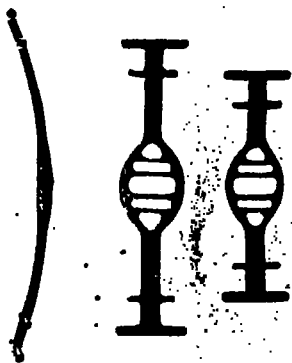


Fig. 10.—Three sizes of ladder. The largest is shown on its side to demonstrate the curve.

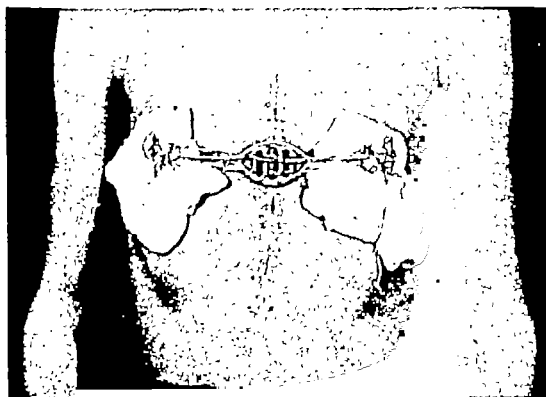


Fig. 11.—Photograph of a patient after operation with ladder in place.

This operation will suffice in most cases. When the only deformity is the prominence of the lower margin of the transverse groove, this can better be corrected by resecting the prominent cartilages or ribs and allowing the chest wall to sink to the level of the groove, rather than by trying to elevate the depression. If there is also some pull on the xiphoid, this bone should be

The external appliance (Fig. 10) was evolved from Brown's ladder and has proved more satisfactory. It is made of duralumin and curved so that the central, fenestrated portion is higher than the ends, thus permitting overcorrection of the sternum. The small

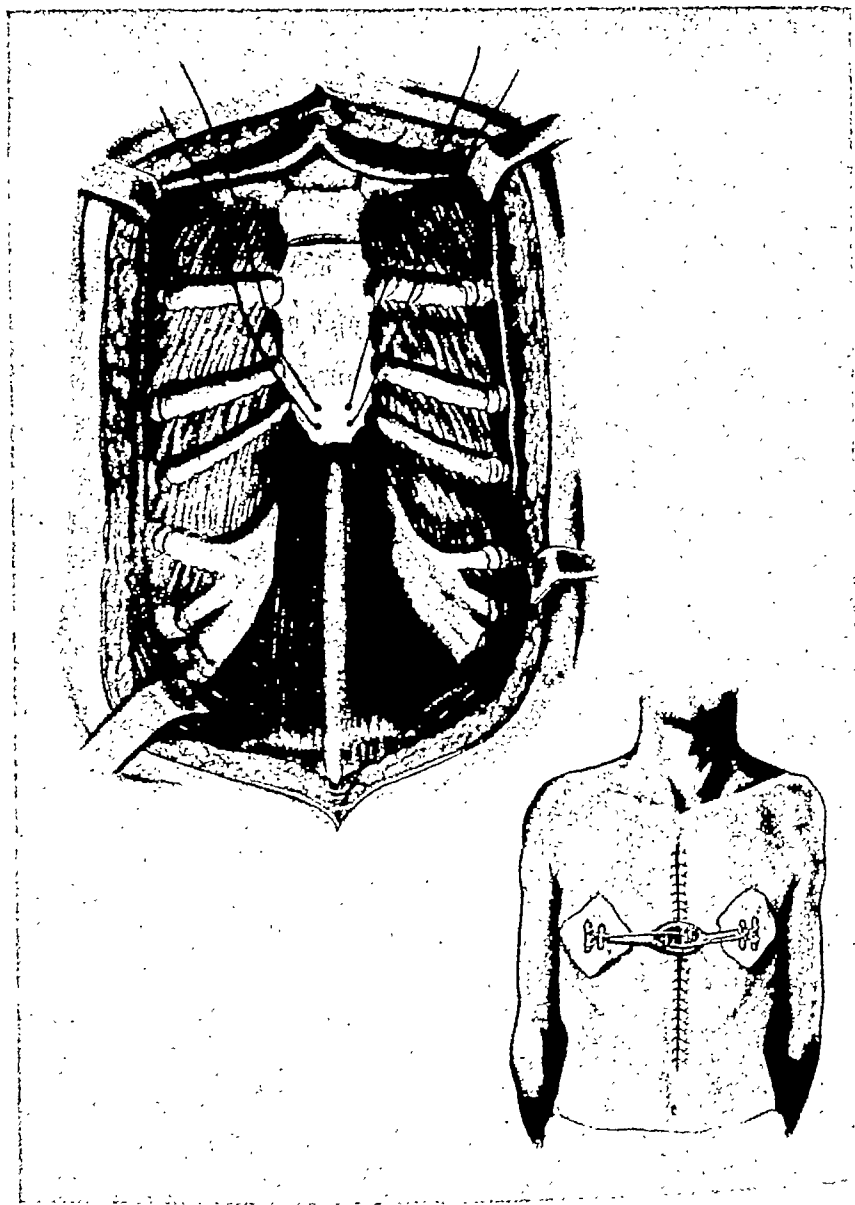


Fig. 9.—Radical operation. The cartilages have been resected, the xiphoid detached and allowed to retract, the wedge osteotomy completed through the outer table across the third sternal segment, and the wires threaded through the drill holes. Insert shows wires brought out through skin on each side of the incision which has been closed, the sternum elevated by the wires and the wires twisted into the ladder.

transverse bars at the ends are used as cleats on which to wind the wires. This permits loosening or tightening of the traction, should either be necessary, and avoids the weakening of the wires which occurs when they are twisted together. The ends of the ladder in contact with the chest wall should be well padded (Fig. 11).

The radical operation performed on patients between the ages of $2\frac{1}{2}$ and $4\frac{1}{2}$ years has given the best results (Fig. 13, *B*). After 5 years of age, and sometimes earlier, permanent changes in the bony structures have developed which cannot be entirely corrected (Fig. 13, *A*). However, operation can be done at any age with the assurance that it will relieve pressure symptoms and probably bring about an 80 per cent correction of the deformity.

The complications characteristic of the operation are not many but are important. The first and most usual is pneumothorax. The perichondrium is so adherent that it is extremely difficult to separate it from the under side of the costal cartilages without tearing the pleura somewhere. The complication is not serious if recognized, even though it may be bilateral. If the opening is too small for the anesthetist to inflate the lung, the air can be aspirated after the wound is closed and there need be no sequelae.



Fig. 13.—*A*, Postoperative result in patient with irreversible changes (Fig. 3, *A*). Sternum fairly well elevated in spite of breaking of wires on second postoperative day but narrow, flattened thorax unchanged. *B*, Postoperative result in child 2 years of age (Fig. 3, *B*). Sternum well elevated and deformity corrected because irreversible changes have not yet occurred.

Breaking of the wires in the early postoperative period is a distressing circumstance with serious possibilities. If it happens in the first few days after operation the resulting paradoxical respiration with the atelectasis and pneumonia which usually follow may be dangerous in the extreme. If it happens later in the course it is not as serious. Replacement of the original silver wire with two of stainless steel has done much to eliminate this complication.

Infection has not been a troublesome problem. In the days before antibiotics we had three instances of serous drainage which persisted for about one

detached as in the operation for infants (Fig. 12). Operations of this sort are done for cosmetic reasons and, as the deformity varies with the individual, should be planned to fit the case.

After operation the patient is given oxygen by means of tent, mask, or catheter. Pneumothorax is a common complication, often unrecognized, and whether or not it is present, the more quiet respiration which oxygen permits is grateful to the patient. Although there should be no great loss of blood, the operation is apt to be long and a blood transfusion during the operation is a good precaution. After operation it is advisable to have a portable roentgenogram taken. Any pneumothorax which it may disclose is corrected at once by aspiration. The position, both in bed and when allowed up, should be one which will correct the exaggerated dorsal curve that is characteristic of the deformity.



Fig. 12.—Individualized operation (Fig. 4, A). Prominences of lower part of groove resected through lateral incisions and xiphoid detached through central incision.

The optimum time for operation is the subject of many queries. In an earlier communication³ we advocated the simple operation in children under 2 years of age but our late results have been disappointing. This is true even when the next pair of cartilages has been resected and the sternum bent up. Follow-up shows that, although the lower end of the sternum is not retracted during inspiration and any pressure on the thoracic organs has been relieved, the deformity still persists to a greater or less degree and has not diminished very much during three years of growth. As the parents bring the child for operation because of the deformity, they consider surgery a failure, even though the child is entirely healthy in other respects and is growing at a normal rate. In 2 patients operated upon at about the age of 1 year the results have been good but in 8 others, somewhat older, they have been equivocal so we prefer to reserve this operation for very young children in whom the deformity is severe and pressure symptoms present.

and I made our report to this organization just ten years ago, and we agree with Dr. Lester's conclusions today that the radical procedure is the desirable one. Of the various methods used we preferred the one we used at that time (slides).

This shows a severe case in a girl. The mobilization of the sternum and costal cartilages is shown here, then the fixation with immobilization in a plaster shell. We have used the Parham-Martin band, because in our experience the wire is likely to cut through; one can use two bands—in this particular case we used one—the plaster shell is prepared beforehand and this is held up with rubber bands. This is the end result.

I would also like to emphasize the importance of long follow-up in these cases, because they do not do as well as we might hope. Recently we had a patient, a man 34 years of age—and his age is of importance—who had a typical chōnē-chondrosternon of the worst type I had seen, and we hoped we might do the same procedure. However, when we got in we found there was so much deformity and so much fixation that mobilization and elevation would not be possible because this extended back so far. We did a procedure which Dr. DeBakey and I, in our presentation ten years ago, said should not be done because it was contrary to all surgical principles. This was first done by Lexer and reported by Hofmeister in 1927. It consisted of turning the sternum and costal cartilages inside out. This has been done several times, in several instances with bad results because of gas bacillus infection. Because of that we meticulously cleaned off all the muscle, leaving only bone and cartilage, and this view shows the result. It was possible to fix the costal cartilages to the side; as a matter of fact we had redundant cartilage. The result has been extremely satisfactory. In the older patient in whom there is marked fixation and marked change, particularly if the deformity extends quite far laterally, I would suggest that the Lexer procedure is a feasible one and will give a good result. But one must remove all the muscle to prevent possible anaerobic infection subsequently. The bone and cartilage will be maintained as a cartilage and bone graft, apparently without any difficulty.

DR. J. E. DAILEY, Houston.—Many techniques have been devised in correcting funnel chests, and I wish to introduce another method in which no casts or any appliances whatsoever are necessary (slides). This could be called, "a rib graft elevation of the sternum." These slides illustrate a 13-year-old girl with quite a marked depression, and the postoperative result. The lateral view shows the marked depression of the sternum. The patient also had electrocardiographic changes and marked cardiac embarrassment. The next slide is a postoperative roentgenogram showing quite a marked improvement in the depression.

The operative procedure in this case consisted in removing a segment of the ninth rib, leaving the periosteum on one surface, then all the cartilages were completely resected on both sides of the sternum leaving the perichondrium. The sternum was elevated. The segment of rib that was removed was then used as a bridge from the second rib on one side to the third rib stump on the other side and wired in place, and the sternum was allowed to rest upon that rib. The perichondrium was then tacked up around the edges of the sternum. The rib placed beneath the sternum, effectively held it in place and the post-operative use of a cast or some suspensory appliance was totally unnecessary.

It is suggested that this technique might be applicable in certain cases of severe crushing injury to the chest in which it might be of value to immobilize the sternum, and counteract paradoxical respiration.

DR. FORRESTER BAINE, Milwaukee.—Along with Dr. Ochsner's remarks about having to turn the sternum when there was such a terrific depression, I have found that by simply splitting the sternum lengthwise with a saw, it can be folded out, and the depression disappears quite well without having to turn it over and it really becomes fixed remarkably so that there is no instability two weeks later. I think that might be simpler than turning it over.

DR. A. L. BROWN, San Francisco.—I do not rise to defend one procedure over another. I think we have come beyond that point. However, I would like to mention our experience in 27 of these cases during the past fourteen years; 12 were the infant type

week but we have yet to encounter a purulent infection. The reason for this may be that the wires act as setons and, of course, penicillin is now used prophylactically.

Edema of the glottis or subglottic region is a complication which must always be considered when endotracheal anesthesia is used in children. Because of the danger of accidental pneumothorax during operation this type of anesthesia must be employed, but in a long operation it certainly predisposes to postoperative tracheal obstruction and a tracheotomy set should always be available near the bedside after operation.

Results have been excellent with respect to the relief of pressure symptoms and in no instance have we had a failure in this category. In certain cases, however, cosmetic results leave something to be desired. This has been discussed with respect to the simple operation. In the older patients the irreversible changes which are prone to develop after the age of 5 years cannot be corrected by elevating the sternum. Flattening and narrowing of the chest due to the downward slant of the ribs will persist. The exaggeration of the normal dorsal vertebral curve can be rectified by corrective exercises if the patient will persist in them. The flare of the lower ribs at the costal arch will give the appearance of a funnel chest after the sternum has been elevated to its normal position and for this reason we have extended the operation to include the cartilages which make up the lower margin of the transverse groove. With this procedure the deformity in this group can be about 80 per cent corrected, depending on the amount of irreversible change, and the patients seem to be satisfied.

The best results have been obtained in the age group between $2\frac{1}{2}$ and $4\frac{1}{2}$ years. There are 5 of these in whom we have done the radical operation and the defect seems to be almost 100 per cent corrected. These cases are all fairly recent and the children have many years of growth ahead of them so no predictions as to the ultimate result should be made at this time.

There have been no deaths among the 31 operative cases.

SUMMARY

It can be said that congenital shortening of the central tendon of the diaphragm produces a variety of deformities in the thoracic cage, the most common and most serious of which is funnel chest. The incapacitating symptoms are due to pressure on the heart. Radical operation in which the costal cartilages are resected, the xiphoid detached from the sternum, and the sternum elevated will relieve the pressure symptoms and bring about a considerable correction of the deformity. The best results are obtained when the operation is done before the age of 5 years.

REFERENCES

1. Ochsner, A., and DeBakey, M.: Chônê-chondrosternon, J. THORACIC SURG. 8: 469, 1939.
2. Brown, A. L.: Pectus Excavatum (Funnel Chest), J. THORACIC SURG. 9: 164, 1939.
3. Lester, C. W.: The Surgical Treatment of Funnel Chest, Ann. Surg. 123: 1003, 1946.

DISCUSSION

DR. ALTON OCHSNER, New Orleans.—This presentation is certainly an interesting one, and Dr. Lester is to be congratulated on the number of cases he has had. Dr. DeBakey

undue traction on the anterior chest wall results and a deformity is produced. This theory is described by me in the March, 1949 issue of *Pediatrics*.

The only diaphragm associated with a congenital chondrosternal depression was seen by me in a 4-week-old suckling puppy. Its anterior segment (derived from the septum transversum) appeared practically absent and its membranous portion instead of being trefoil consisted of two leaves which were fused anteriorly and attached to the xiphoid-gladiolar junction.

I found the anatomy of the diaphragm quite different in a 14-month-old infant with a congenital chondrosternal prominence (pigeon chest). The membranous portion was in the shape of an inverted letter "V." The anterior portion contained few thin muscle fibers compared to the strong muscle fibers of the lateral portions. The contraction of such a diaphragm apparently produces this type of deformed chest, namely, a round full "infantile" upper half which forms the prominence and a comparatively retracted lower half of its anterior wall.

As a result of these observations and studies, I feel that congenital deformities of the anterior chest wall result from the contractions of a congenital abnormally developed diaphragm.

In patients with congenital chondrosternal depressions, I employ an inverted "Y" incision. For the correction and maintenance of the sternum I suture a free cartilage graft, taken from the costal margins, under the end of the elevated sternum to the ends of the fifth cartilage.

DR. PAUL HOLINGER, Chicago.—The problem of funnel chest is of interest to the laryngologist as well as the thoracic surgeon. Any chronic laryngeal obstruction will result in temporary and possibly permanent funnel chest deformity. This is seen in infants and children with laryngeal papillomatosis or with so-called "congenital laryngeal stridor." In the latter condition, the larynx is soft, flaccid, and slightly edematous and on each inspiration the arytenoids, aryepiglottic folds, and curled epiglottis are sucked into the glottic lumen to cause obstruction. This is often severe enough to result in extreme sternal retraction with the epigastrium drawn back to the spinal column on inspiration. It is certainly conceivable that the shortening of the ligament of the diaphragm, which is considered an etiologic factor of funnel chest, is the result rather than the cause of the retraction of the sternum. The retraction in congenital stridor is usually present at birth and increases in severity with an alarming increase in the loud stridorous sound on inspiration until the infant reaches 10 to 12 months of age. Thereafter the noisy breathing gradually decreases and at 2 or 3 years of age breathing is entirely normal; the larynx has assumed its normal rigidity and stays patent on inspiration. But, in the severe cases, the infant has developed a chest deformity with flaring of the ribs and, to a greater or lesser degree a funnel chest.

The extreme deformities presented by Dr. Lester certainly required operative repair. But indications for surgery in some of the operated infants shown by discussants might be questioned, especially when expiration films of the chest showed a normal anteroposterior diameter of these chests. In such cases, the deformity could hardly be considered as due to a shortened diaphragmatic ligament, since then the deformity would be evident on expiration as well as on inspiration, although possibly not to the same degree. I think, therefore, that before operating any infant for this chest deformity, a careful study of the larynx should be made by direct laryngoscopy; and that the likelihood of a spontaneous disappearance of the deformity as the infant reaches $2\frac{1}{2}$ to 3 years of age is great. The indication for surgical intervention might better be the degree of displacement of mediastinal structures rather than the degree of substernal retraction on inspiration.

DR. JEROME HEAD, Chicago.—We have recently had a case of this type which we handled in a different manner. It seemed to us the deformity could be corrected best by applying force in the opposite direction from the force which had been applied to create the deformity, and doing this without resection of cartilage or transection of the sternum.

procedure, and by this we mean 2 years of age or under; when we speak of the adult type it is over that age. In the infant type if they are done when the development of the defect is still going on you get a result. If you do it after that you get no result.

So far as technique goes, we do not use any support at all. The whole trick, so far as I am concerned, is in freely developing motion of the sternum, overcorrecting the defect, and the sternum sits up by itself. Then if you wish you can put an extra suture in any one of the rib stumps that seems to be pretty close to the sternum; it will stay up whether you put that in or not. We have reached the point where we are trying to evaluate when one should do the operation. People come for two reasons; one is for the cosmetic result, and that is important; but the other is, for a person without symptoms with the defect or a child who is developing the defect, should you advise operation? The end results, from the cosmetic viewpoint, have been very good in children and fair in adults. I would say about 75 per cent in the adults. We have had no mortality in the 27 cases. But from the functional standpoint, where the patient presents himself with symptoms, the end results have been poor, uniformly poor, and that has led us to undertake cardiorespiratory studies on these patients before surgery and as soon as they are seen. In the early cases this is difficult but as they get along in age you can make good studies and, surprisingly, the adult has some cardiorespiratory signs before he is conscious of them (slides).

This is an outline of the studies we are now making. This child had lung on only one side. Postoperatively all get an immediate response in the breathing reserve, and the breathing capacity is immediately increased. This shows an adult of about 33 years of age, who started with a very fine vital capacity, very marked deformity. We do not consider that vital capacity means anything. The maximum breathing capacity before operation was 15, and 32 immediately after. However, they drop as months go on.

This slide shows the development of the pectus in a child; you see what is going on. There is neuromuscular imbalance which gradually causes permanent shortening in the anteroposterior diameter of the diaphragm.

At present we are trying to determine when operation should be advised and as it stands now, we think you cannot do it early enough.

DR. HENRY A. BRODKIN, Newark, N. J.—I am of the opinion that rickets plays no part in the formation of anterior deformities of the chest, commonly called funnel chest, pigeon breast, and Harrison's grooves, which I had the temerity to name congenital chondrosternal depression, congenital chondrosternal prominence, and congenital costochondral depressions, respectively. I believe these deformities result from the altered contractions of an abnormally developed diaphragm.

The importance of the diaphragm in respiration is realized when one reflects on the fact that a young animal will die of respiratory failure when both its phrenic nerves are severed. I believe further that it is chiefly the influence of the action of the diaphragm that changes the contour of the infantile round chest to the elliptical "adult" contour at the end of the first year.

After observing the synchronous retractions in the anterior chest wall during inspiration with the contraction of the diaphragm in two infants, one with a congenital chondrosternal depression (funnel chest) and the other with a congenital chondrosternal prominence (pigeon chest), a phrenic nerve of each was injected with 1 per cent Novocain. The retraction in each deformity ceased on the side injected. This was strong evidence to me that the deformity was produced by the contractions of an abnormal diaphragm.

After reviewing the embryology of the diaphragm and studying the diaphragms of various animals, I came to the conclusion that a normal diaphragm has a definite anatomic structure with a definite structural ratio between its circumferential radial musculature and its trefoil membranous portion. It consists of four fused segments, an anterior, two lateral, and a posterior, each separated by a fibrous raphe, derived respectively from the anterior septum transversum, the two lateral pleuroperitoneal membranes, and the dorsal mesentery. Contraction of a normal diaphragm exerts its traction on the membranous portion. If the membranous portion extends to the sternum or if the normal structural ratio is not present,

AN EXPERIMENTAL STUDY OF THE BLOOD SUPPLY OF THE ESOPHAGUS AND ITS RELATION TO ESOPHAGEAL RESECTION AND ANASTOMOSES

JOHN L. SHEK, M.D. (BY INVITATION), CARLOS A. PRIETTO, M.D.; M.S. (SURGERY)
(BY INVITATION), W. M. TUTTLE, M.D., AND E. J. O'BRIEN, M.D.
DETROIT, MICH.

INTRODUCTION

THIS series of experiments was ventured upon to ascertain whether or not the blood supply of the esophagus of the dog is adequate to tolerate a resection with end-to-end anastomosis.

METHODS OF SURGERY

Thirty-two dogs, whose average weight was 15 pounds, were used in these experiments. A preoperative dose of morphine sulfate, 0.09 Gm., and atropine sulfate, 0.004 Gm., was given one hour before surgery. Positive pressure endotracheal ether and air anesthesia was used. The left pleural cavity was entered through the bed of the sixth rib, which had been resected.

In these experiments, varying lengths of the esophagus were resected and it was denied of varying amounts of blood supply. In all experiments the resected ends of esophagus were reunited by single interrupted horizontal mattress sutures of 00000 silk in two layers. The esophagus was then replaced in its mediastinal bed, except in two dogs of the first experiment. These had the esophagus transplanted anterior to the aortic arch.

The mediastinal pleura was not closed. The lungs were re-expanded, 100,000 units of penicillin were left in the pleural cavity, and the thoracic wall was closed in layers with interrupted sutures of cotton.

Immediately postoperatively, the animals were given 100,000 units of penicillin intramuscularly. No more penicillin was administered.

Food was denied the animals for the first twenty-four postoperative hours. During the next four days a diluted mixture of the ordinary dog food was given. Following this, the animals were given a regular diet of commercial dog meal, mixed with water.

METHODS OF ARTERIOGRAPHY

For all arteriograms, the thoracotomy wound was reopened, and the pleural cavity entered, using the aforementioned surgical technique. The lung was packed away to visualize the esophagus. A right rectus abdominal incision was made and the diaphragm divided. Through this thoracoabdominal incision, ample delivery and mobilization of the stomach was achieved. It was essential that the animal remain alive, for in these experiments, the propulsion of the opaque media was accomplished by the animal's arterial pressure.

The media used in the first arteriogram was a mixture of 20 per cent bismuth oxychloride in 1 per cent starch solution. The bismuth was easily seen through the arterial walls. In all other experiments a mixture of 18 per cent bismuth

From the Department of Surgery, Wayne University College of Medicine, and the Surgical Laboratories of the W. H. Maybury Sanatorium (City of Detroit Department of Health).

Read at the Twenty-ninth Annual Meeting of The American Association for Thoracic Surgery, New Orleans, La., March 29-31, 1949.

Transecting the sternum and removing the cartilage tend to elevate the whole deformity rather than correct it, and correct the deformity of the ribs which had been occasioned by depression of the sternum. Therefore we made an incision and removed the xiphoid cartilage and simply put a hook under the lower end of the sternum, out through the skin, and applied traction to it. To hold the hook in place we put a stainless steel wire beneath the sternum and fastened that around the hook to hold it. After ten days we removed the hook from beneath the end of the sternum, and since then have applied traction early by rubber bands attached to the wire which runs beneath the sternum (slides). We did not use the bridge across the chest as we did not want to apply counter-pressure to the ribs, and felt if we were pulling the sternum out we would also be pressing the ribs back. In this case the heart had escaped to the right, which is rather unusual. There is a residual deformity on the left, and we plan to put wire there and pull this out in the same manner. The principle is the same as dentists use in correcting malalignment in teeth.

DR. LESTER.—I appreciate the discussion which has been evoked. Dr. Ochsner's plaster shell for correction I think is admirable, and one can certainly get good correction by taking the sternum out and turning it over. I should say it would take some time to heal, during which it keeps the patient in bed in a plaster shell; and the same thing applies to Dr. Head's appliance. The results are good, there is no question, but the children with the external appliance which Dr. Brown has used and which he has given up, are allowed up and out of bed about the third day, and are running around the wards without any disability.

This is Dr. Brown's baby, really. It is his operation, and I am interested in his remarks on giving up the use of the ladder. We find that when the wires have broken we get a great deal of paradoxical motion. In the 19-year-old boy I showed this was extreme and had us much worried for two weeks. Every time he breathed the sternum flopped back and forth like a flag in the wind. In the obstruction situation that Dr. Holinger mentioned, that is in line with the observation made by Dr. Emmett Holt, Jr., Professor of Pediatrics at New York University. He has been interested in these cases and he suggested some sort of laryngeal obstruction as a possible cause of the trouble, and he delights in calling me up to see children that have laryngeal obstruction with retraction of the sternum during forced inspiration. Of course, you get that in any type of laryngeal obstruction. That is one of the cardinal signs of laryngeal obstruction which may call for tracheotomy. I think, however, that you cannot correct a true funnel chest by correcting that obstruction, and many do not have an obstruction at all. However, when a child has large tonsils or other evidence of obstruction to his upper respiratory tract, we always correct that before we try to correct the chest.

In these freed areas, portions of esophagus ranging in lengths from 3 to 5 cm. (that is, approximately one-seventh to one-fourth of the entire thoracic length), were resected. The esophagi of two of the fifteen dogs were resected at the level of the aortic arch. The esophagus was transplanted from behind the arch and the anastomosis was made anteriorly.

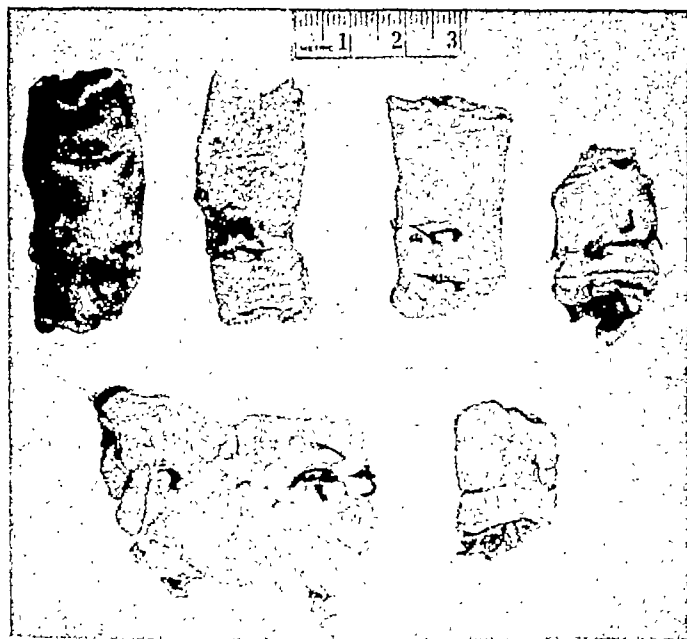


Fig. 2.—Autopsy specimen of esophagi more than two months after resection and anastomosis. First two, upper left, show muscularis surfaces. Others show mucosal surface after specimen is turned inside out. (Experiment 1.)

Results.—The animals recovered. Seven dogs were noticed to retch during the second week whenever solid food was eaten too rapidly. Esophagoscopy was done, and revealed no visible stricture. Fourteen dogs survived the operation for longer than two months and were then sacrificed. One dog died on the twenty-second postoperative day. An autopsy on this animal revealed patchy consolidation of the left lung due to bronchopneumonia. The esophagus had a well-healed suture line around which there was no evidence of infection. In the fourteen dogs sacrificed, the anastomosis in each instance was perfectly healed and there was no evidence of stricture (Fig. 2).

oxychloride in 35 per cent Diodrast was used. This media was easier to work with as it flowed more easily through a 25 gauge hypodermic needle. The amounts of media injected varied from 8 to 30 c.c.

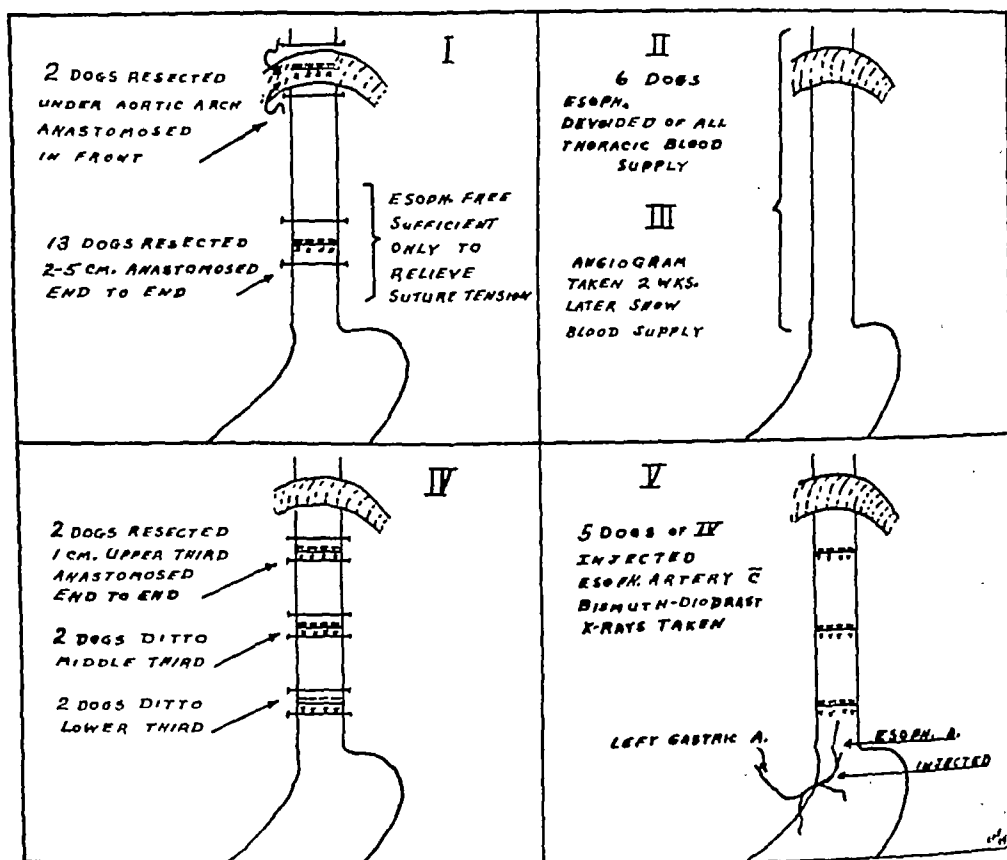


Fig. 1.—Diagrammatic sketch. I refers to Experiment I; II and III refer to Experiments II and II, A; IV refers to Experiment III, and V refers to Experiment III, A.

When the left gastric artery was injected, all vessels to the right were ligated and the right half of the stomach was clamped to prevent unnecessary filling in this direction. When the right gastric or right gastroepiploic arteries were injected, the duodenum was clamped and the vessels to the omentum were ligated. After introduction of the needle into the artery, a ligature around the artery held the ensheathed needle in place. The injection was made slowly in order to allow the blood to carry the media and to avoid vessel rupture.

After all arteriograms, the stomach and esophagus were removed as a specimen. X-ray films were then made. The dog was sacrificed and if previously operated, an autopsy performed.

EXPERIMENTS

I. To Determine the Feasibility of an End-to-end Anastomosis of the Thoracic Esophagus (Fifteen Dogs Used)

Varying lengths in the esophagus were freed from their blood supply in that area which reached from the diaphragm to the arch of the aorta (Fig. 1).

*III. To Determine the Adequacy of the Blood Supply From the Left Gastric Artery in Healing an Anastomosis Done After Resection at Different Levels in the Esophagus Devoid of All Thoracic Blood Supply
(Six Dogs Used)*

In all animals in this experiment, the esophagus was deprived of its thoracic blood supply. In each pair of dogs 1 cm. of the lower, the middle, and the upper third of the esophagus was resected. The anastomosis was made as before.

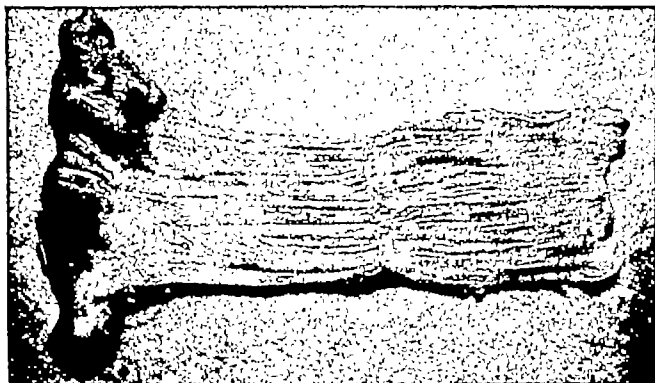


Fig. 4.—Experiment III. Anastomotic line on mucosal surface three weeks postoperative.



Fig. 5.—Experiment III, A. Arteriogram of esophagi, three weeks after resection and anastomosis. Injection media seen carried to level of anastomosis and beyond.

*II. To Determine the Viability of the Esophagus Devoid of
Its Entire Thoracic Blood Supply
(Six Dogs Used)*

In this experiment, the entire length of the esophagus was freed and all its thoracic blood supply was severed (that is, those vessels from the bronchial and intercostal arteries, as well as those from the thoracic aorta and aortic arch).

Results.—All six dogs survived the operation, and five lived and ate well for a period longer than fourteen days, after which they were used for arteriogram studies. The sixth animal died on the fourth postoperative day and an autopsy revealed a dilated right heart. The esophagus was viable. At autopsy, done subsequent to the following experiment, the esophagi of the remaining five animals appeared normal.

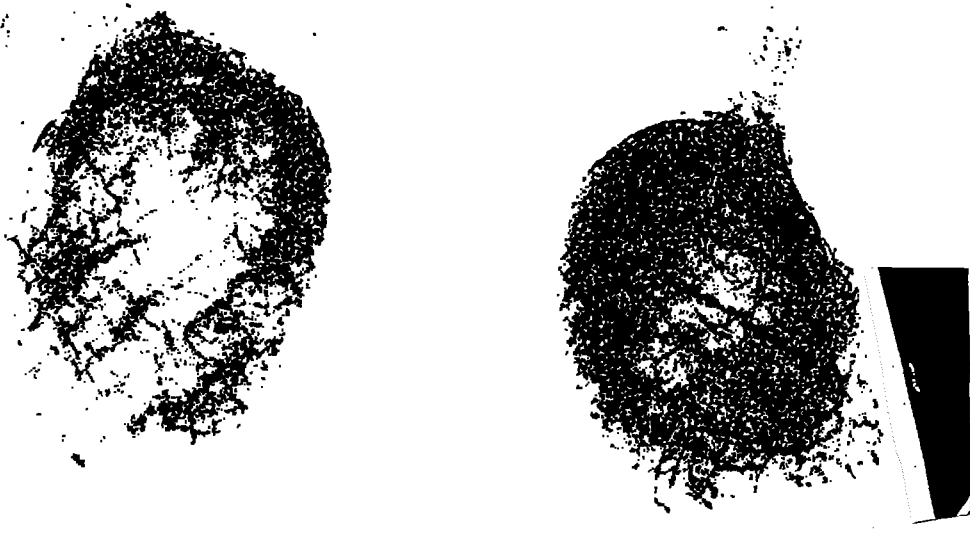


Fig. 3.—Experiment II.A. Arteriogram showing injection media carried to upper one-fourth of esophagus, after being injected into the right gastric artery.

A. To Determine by Arteriogram How Much of This Esophagus Is Supplied by Branches From the Left Gastric Artery (Same Dogs Used as Were Used in Previous Experiment).—Using the aforementioned arteriography technique, the left gastric artery was injected with approximately 10 c.c. of the prepared media.

Results.—The injection media was carried from the gastric arteries into the esophagus and passed upward to approximately its upper one-fourth (Fig. 3).

thoracic esophagus was freed as before. The diaphragm was opened and the stomach delivered into the thoracic cavity. The left gastric, left gastroepiploic, and short gastric arteries were ligated and severed. Immediately following this, the right gastroepiploic artery was injected with 20 to 30 c.c. of the prepared bismuth and Diodrast mixture. The esophagus and stomach were removed and an x-ray film made.

Results.—The x-ray films revealed that the injection media has been carried into the cervical esophagus (Figs. 6 and 7).

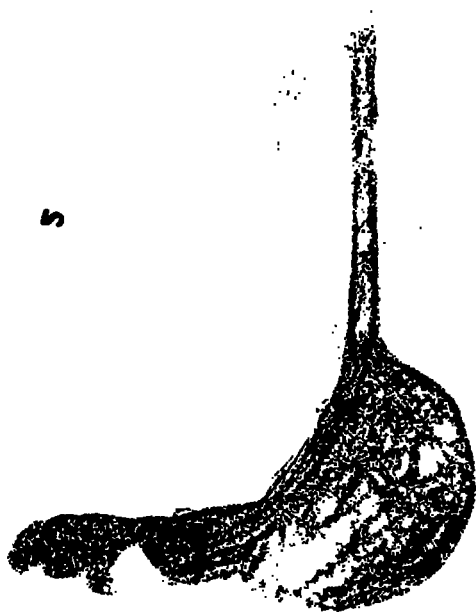


Fig. 7.—Experiment IV. Injection media seen carried into cervical esophagus from the right gastroepiploic artery.

V. *A Study of Healing of an Esophagus Resected and Anastomosed After It Is Devoid of All Thoracic Blood Supply and the Left Gastric, Left Gastroepiploic, and Short Gastrics Are Divided (Two Dogs Used)*

In this experiment, the esophagus was freed of all its blood supply as before, in addition to which the left gastric, left gastroepiploic, and short gastric arteries were ligated and severed. The esophagus was then resected of 1 cm. in the middle third and anastomosed end-to-end.

Results.—All six dogs survived the operation. Five lived for three weeks, at which time they were reoperated upon for the next experiment. The sixth is still alive after eight weeks. Subsequent to the following experiment, autopsy of the five dogs revealed good healing of the esophagus and no evidence of stricture (Fig. 4).

A. Arteriogram Study of the Esophagus Three Weeks After It Has Been Resected at Different Levels and Anastomosed When Devoid of All Thoracic Blood Supply (Same Five Dogs Used as Were Used in Previous Experiment).—In this experiment, the left gastric artery was injected with 8 to 16 c.c. of the prepared media, using the previously described technique.

Results.—Arteriograms revealed clearly the adequacy of the blood supply of these esophagi as shown by the fact that the injection media was carried to the line of anastomosis or beyond (Fig. 5).

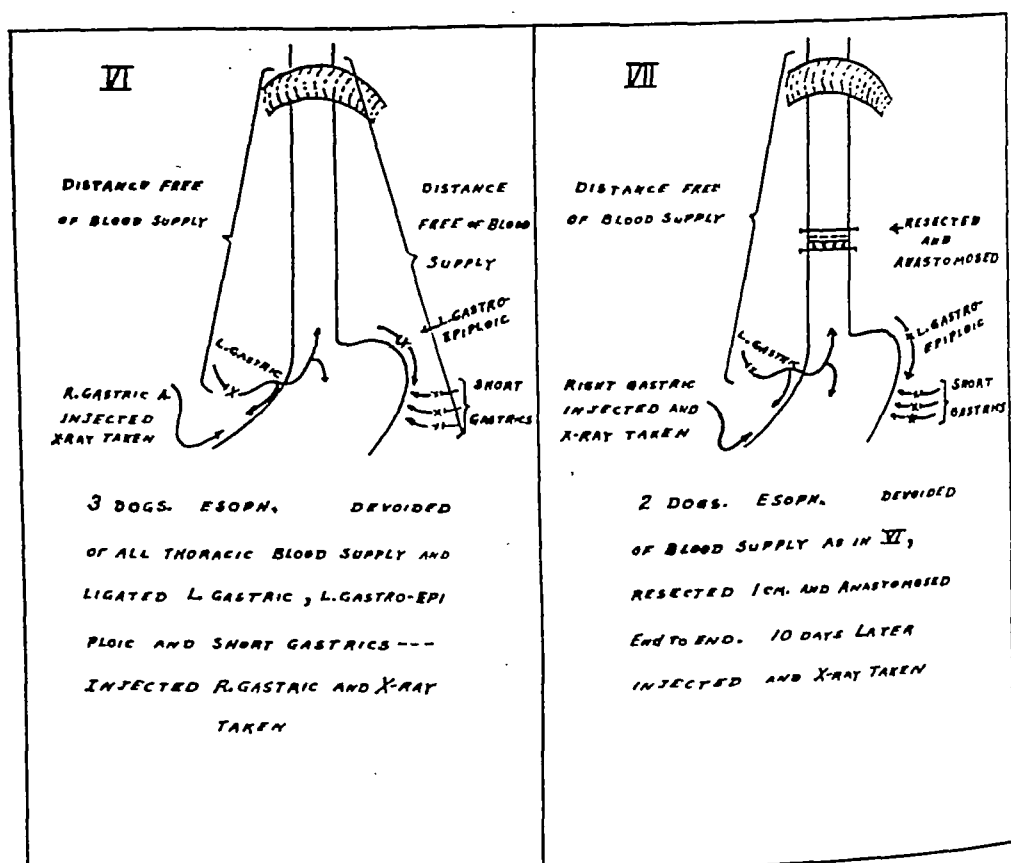


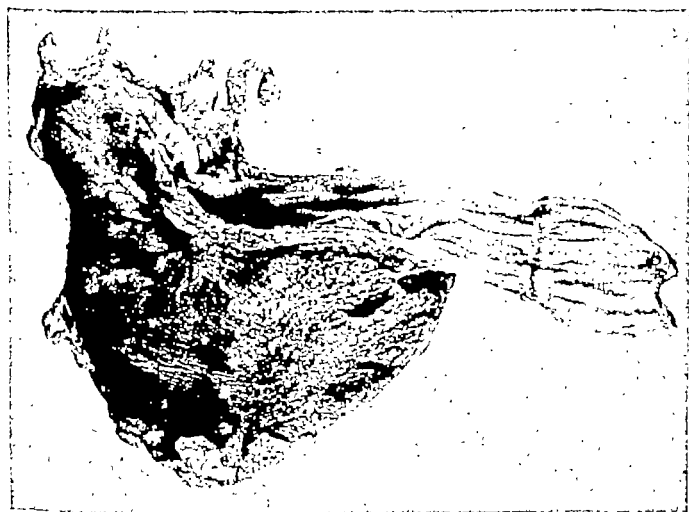
Fig. 6.—Diagrammatic sketch. VI refers to Experiment IV and VII refers to Experiments V and V, A.

IV. Arteriogram Study of the Esophagus After It Is Devoid of All Thoracic Blood Supply and the Left Gastric, Left Gastroepiploic, and Short Gastric Arteries Are Divided (Three Dogs Used)

This experiment was done to determine whether or not the blood from the right gastric artery could reach the esophagus by anastomotic channels. The



A.



B.

FIG. 9.—Experiment V. A, Second specimen. Arrow points to anastomotic line on muscularis surface, ten days postoperative. B, Second specimen. Anastomotic line on mucosal surface, ten days postoperative.



A.



B.

Fig. 8.—Experiment V. A, Arrow points to anastomotic line on muscularis surface ten days postoperative. B, Anastomosis line on mucosal surface ten days postoperative. (Note ascaris lumbricoides at prepyloric region.)

Interestingly enough, in this entire series of experiments no animal suffered immediate postoperative death. Two dogs died postoperatively, one on the fourth day from apparent right heart failure, and the second on the twenty-second postoperative day from a bronchopneumonia. In each instance, the esophagus was intact and no mediastinitis was present.

In these animals it has been well demonstrated that not only can the entire thoracic esophagus be deprived of its blood supply and survive well, but at the same time portions can be resected and the esophagus reunited by end-to-end anastomosis with perfect healing. This would tend to prove that the arterial supply from the gastric arteries is sufficient to nourish the entire esophagus.

Arteriograms made on animals whose entire thoracic and upper gastric blood supply had been ligated revealed the fact that the right gastric artery can, through its anastomotic connections, supply the esophagus as high as its cervical segment.

Criticism may be directed against these experiments in that no long-term animals (the longest is two months) have been reported. The fear may be raised that after a period of months, stricture may occur at the point of anastomosis. It will be noted from a perusal of Figs. 2, 4, and 7, that no apparent narrowing of the esophagus is evident. Experiments are in progress to clarify this point.

Antibiotics were used sparingly in these studies. This was done to test more drastically the adequacy of the blood supply.

CONCLUSIONS

1. Local resection of the esophagus with end-to-end anastomosis has been successful.

2. The entire esophagus can be freed of its thoracic blood supply and still remain viable.

3. When the entire esophagus is freed of its thoracic blood supply and is resected and anastomosis made, it heals well.

4. It has been demonstrated by arteriograms that the esophageal blood supply which is derived from the stomach, even though all the arteries except the right gastric are severed, is sufficient to keep the esophagus viable and heal an anastomosis.

(See discussion on page 537.)

Results.—Both dogs survived until sacrificed ten days later for the following experiment. Autopsy subsequent to the following experiment revealed good healing of the suture line and no evidence of stricture (Figs. 8, *A* and *B*, and 9, *A* and *B*).

A. Arteriogram Study of This (V.) Experiment (Same Two Dogs Used as in Previous Experiment).—The previous two dogs were reoperated and the right gastroepiploic or right gastric artery was injected with the prepared media.

Results.—The injection in one dog was poor, due to technical difficulties. The other, however, showed the injection media had reached the line of anastomosis (Fig. 10).



Fig. 10.—Experiment V,A. Arteriogram showing injection media carried to suture line.

DISCUSSION

These experiments reveal the fact that apparently the blood supply is much more adequate than had been previously supposed, both by the actual clinical healing in the experimental animal, and by the arteriographic studies of the esophageal blood supply.

It will be noted that in each instance where a portion of the esophagus was resected and then an anastomosis was made, healing occurred readily and well.

OPERATIVE PROCEDURE

The first type of procedure was used in three patients. The chest was entered through the bed of the eighth rib, the esophagus was freed from its bed, and the area of stricture was identified. The normal esophagus above and below the narrowed area was held upward on rubber drain slings. A triangular wedge of esophagus was then removed in such a manner as to preserve a small strand of esophageal tissue posteriorly to act as an anchor. The cut ends were then joined by interrupted sutures of fine silk, one layer being placed in the mucous membrane and a second layer in the muscularis and adventitia. The esophagus was then replaced in the mediastinum. The mediastinal pleura was lightly closed.

The second approach to this problem was used in two patients. In these patients the mediastinum was approached as in the first group. The esophagus was raised from the mediastinum and after the area of stricture had been identified it was completely resected. The lumen was re-established by an end-to-end anastomosis using fine interrupted silk sutures in two layers as in the first group with the exception that the resection was complete.

RESULTS

The long-term results from a standpoint of patient survival were not good. Three of the five patients are now dead. However, in none of the three who died at intervals of from a week to three months following surgery was there any evidence that the anastomotic line had broken down.

CASE REPORTS

CASE 1.—J. H., a Negro boy, 7 years of age, was delivered normally in 1941. It was noticed when the boy began to take semisolid food at the age of 9 to 10 months that some trouble was experienced in swallowing such a diet. This difficulty progressed and at the age of 2 years a gastrostomy was done. From that time until 1946, the esophagus was dilated both through the gastrostomy and through the upper esophagus. During 1945 and 1946, it appeared that he was able to eat with moderate ease, and the gastrostomy was allowed to close.

He apparently ate well but continued to show evidence of a persistent anemia. Numerous and careful studies of the blood failed to reveal the exact nature of his anemia. In March, 1948, he was again admitted to The Children's Hospital of Michigan. At this time there was evidence of a severe secondary anemia and dysphagia. X-ray studies revealed a definite stricture of the esophagus in its lower one-third. Esophagoscopy was done and there was a weblike stricture seen across the lower one-third of the esophagus. A filling defect was seen in the stomach. There was occult blood in the stool.

It was decided to explore the esophagus and stomach transthoracically. The patient's blood was brought to normal and on April 12, 1948, the operation was performed.

The chest was opened and the esophagus raised from its bed. The area of stricture was palpated and appeared approximately 2 cm. in length. It was decided to resect it locally and not to enter the stomach as it was felt that the filling defect in the stomach was due to the deformity caused by the previous gastrostomy.

Therefore, a wedge which included the stricture except for its extreme posterior portion was removed from each side. This left a narrow, 2 mm. band posteriorly, as an anchor. The esophagus was reunited with two layers of fine silk.

The postoperative course was uneventful and the patient was taking a full diet on the eighth postoperative day.

THE TREATMENT OF SHORT ESOPHAGEAL STRICTURES BY RESECTION AND END-TO-END ANASTOMOSIS

W. M. TUTTLE, M.D., AND J. C. DAY, M.D.

DETROIT, MICH.

IT HAS long been believed that resection of portions of the esophagus and a re-establishment of the lumen by an end-to-end anastomosis was impossible. The lack of a supposedly adequate blood supply and the absence of a serosal coat reportedly would cause the anastomotic line to break down. It was for this reason that Adams and Phemister¹ and later Sweet³ and Garlock² brought the stomach into the thoracic cavity and joined it to the esophagus above the resected tumor. The use of esophagogastrostomy to re-establish the continuity of alimentary tract following esophagectomy has proved a most useful and adequate procedure.

Often times in the course of resecting a long length of esophagus to overcome a very short area of stricture, we have been impressed by the magnitude of the surgical procedure used to correct the small but important defect. The thought arose, would not a direct attack with subsequent end-to-end closure be feasible, especially in view of the now prevalent view that healing in the esophagus takes place by proper approximation of the mucosal layer. Good union and healing without stricture is seen frequently when large diverticula of the esophagus are removed in a one-stage operation. If healing occurs in such defects, which are often large, why should it not occur in end-to-end procedures?

We were encouraged in this thought by the previously mentioned observations, by the adequate healing seen in the operative procedure of cardioplasty for achalasia, where often large areas of an exceedingly poor esophageal wall are joined to the stomach with gratifying results, and by the results of experiments carried out in our own laboratory.

At the onset we were not bold enough to resect completely the entire annular stricture and then directly reunite the superior and inferior aspects of normal esophagus. In patients previously operated upon who are not included in this group, the stricture was cut through longitudinally and the closure was made in two layers in a horizontal direction. These wounds healed well and function was restored. Such results encouraged us to proceed with the two types of operative procedures to be reported here.

MATERIAL

This report is based on the results experienced in five patients with esophageal stricture who ranged in age from 1 to 70 years. The areas of stricture varied in length from 1 to 2½ cm. The causative agent in none of the patients was clear, but in the two children it was obviously congenital, for symptoms of dysphagia had been present since birth.

From the Department of Surgery, Wayne University College of Medicine.
Read at the Twenty-ninth Annual Meeting of The American Association for Thoracic Surgery, New Orleans, La., March 29-31, 1949.

It is unfortunate that the fatality figures here reported are as high as they are, but it is gratifying that necropsy was obtained in two and that in each instance there was evidence of good healing.

DISCUSSION

The procedure described in this paper will not be of use in malignant lesions of the esophagus. In such patients the amount of esophagus to be resected would be too extensive and the procedure of esophagogastrostomy is the only feasible approach. However, it does appear to have a definite place in the short esophageal stricture. It appears from the foregoing results that blood supply of the esophagus is adequate to withstand this operative procedure and that healing will occur. Recently we have carried out a group of experiments in our laboratory which show that in the dog at least the esophageal blood supply is adequate when all the esophageal and gastric vessels with the exception of the right gastric are tied.

CONCLUSION

The resection of a short esophageal stricture with an end-to-end anastomotic repair is feasible.

REFERENCES

1. Adams, W. E., and Phemister, D. B.: Carcinoma of the Lower Thoracic Esophagus; Report of a Successful Resection and Esophagogastrostomy, *J. THORACIC SURG.* 7: 621-632, 1938.
2. Garlock, John H.: The Surgical Treatment of Carcinoma of the Thoracic Esophagus; With a Report of Three Successful Cases, *Surg., Gynec. & Obst.* 66: 534-548, 1938.
3. Sweet, Richard H.: Surgical Management of Carcinoma of the Mid-thoracic Esophagus. Preliminary Report, *New England J. Med.* 223: 1-7, 1945.

DISCUSSION ON "AN EXPERIMENTAL STUDY OF THE BLOOD SUPPLY OF THE ESOPHAGUS AND ITS RELATION TO ESOPHAGEAL RESECTION AND ANASTOMOSIS" BY DR.

JOHN L. SHEK, DR. CARLOS A. PRIETTO, DR. W. M. TUTTLE, AND DR. E.

J. O'BRIEN*; AND "THE TREATMENT OF SHORT ESOPHAGEAL

STRICTURES BY RESECTION AND END-TO-END ANASTOMOSIS"

BY DR. W. M. TUTTLE AND DR. J. C. DAY

DR. JOHN R. PAINE, Buffalo, N. Y.—Some of us at Buffalo General Hospital are engaged in a series of experiments very similar to those shown in the first paper. Our experience up to the present time confirms entirely the results Dr. Shek has shown you. We have carried the devascularization of the upper gastrointestinal tract to the extent of freeing entirely from its blood supply the cervical esophagus, the esophagus in the chest, and the stomach down as far as the right gastric artery and the right gastroepiploic artery. I would like to show a few slides of some of the specimens we have obtained. (Slides.)

In three dogs the entire thoracic esophagus was freed from its bed and its segmental blood supply severed. The esophagus was then cut transversely in middle third and re-sutured. No gangrene resulted. Healing of the anastomosis was satisfactory.

In two animals treated similarly, the anastomosis was made at about the junction of the lower and middle thirds of the esophagus. In two additional animals the anastomosis was placed at the junction of the upper and middle thirds. Healing was satisfactory in all these animals without gangrene.

*See page 523 for article by Shek, Prietto, Tuttle, and O'Brien.

He left the hospital on the fourteenth postoperative day. He was readmitted to the hospital in July, 1948. The red blood count was again approximately 2,500,000 and there was again occult blood in the stool. In order to bring his blood again to normal a transfusion was given. Following this his temperature rose to 107° F., he became stuporous and anuric and died two days later in coma.

An autopsy was obtained. This revealed the esophagus well healed and the lumen adequate. There was no true area of bleeding found in the entire gastrointestinal tract.

The kidneys revealed some cloudy swelling but they were not the true picture of the anuric kidney seen after a transfusion reaction.

At the time this child had received his blood there had been some trouble in the local blood bank with blood which had been contaminated with a coliform bacillus. Such an organism was found in the heart blood and it was felt that the blood given may have contained this organism.

This child probably represents one of those persons who bleeds from the gastrointestinal tract but in whom no source of the bleeding is ever found.

The autopsy did reveal the esophageal stricture adequately cared for.

CASE 2.—P. M. was born in late 1946. Soon after birth some difficulty in swallowing was noted. She did, however, gain weight normally on a liquid diet and experienced no real trouble until solid foods were given. At approximately 18 months of age she entered the St. Mary's Hospital, Detroit. X-ray studies of the esophagus revealed a high-grade stricture in the lower one-third.

The child was well formed and normally developed. Laboratory findings with the exception of a mild secondary anemia were normal. Blood was given and the child was taken to surgery for correction of the esophageal lesion. She was given an anesthetic of ether and oxygen. Before the endotracheal tube was introduced, the child became pale and the pulse weak and thready. The anesthetic was stopped and the child recovered. The operative procedure was postponed and the child returned to the ward.

Five days later, at which time the child was perfectly normal, she was again taken to the operating room. This time anesthesia was induced without trouble. The chest was opened by resecting a portion of the left eighth rib. The mediastinum was opened and the esophagus raised up, and the stricture was identified.

The esophagus was resected above and below the stricture and the cut ends were united with interrupted silk sutures in two layers. A tube was left in the chest and connected to a closed drainage bottle.

The postoperative course was uneventful. Liquids were started on the second postoperative day. These were increased and on the fifth postoperative day the diet was changed to a normal one for the child.

On the evening of the seventh postoperative day, while the nurse was feeding her, the child suddenly and without warning died.

An autopsy was performed. No cause of death was found. The esophagus was well healed and there was no evidence of leakage.

Whether this was one of those peculiar instances of sudden death in children, the cause of which is never clear, cannot be said. It can be stated that the autopsy, except for healing of the esophagus, was not informative.

The patient had a well-healed esophagus at one week and there was no evidence of leakage. The child had been taking a normal diet for two days at the time of her sudden death.

CASE 3.—Another fatality occurred in a 70-year-old man who died approximately ten days after surgery. Death was due to a cerebral accident and no autopsy was obtained. However, esophageal function was good and there was no clinical evidence of leakage.

CASES 4 AND 5.—The two remaining patients have good esophageal function at this time and had a smooth and uneventful postoperative course. Their esophageal function remains adequate at this time, which is approximately eighteen months following surgery.

DR. JAMES M. MASON III, Birmingham.—Dr. Tuttle mentioned the technique of longitudinal incision with plastic reconstruction of short benign esophageal strictures. This technique, we believe, will have a place in some such cases. I would like to present the following case history. (Slides.)

Jefferson-Hillman Hospital, No. 40188.—L. N., colored man, married, aged 46 years, was admitted to the hospital on Jan. 24, 1947, with a stricture high in the cervical esophagus which followed the accidental swallowing of lye four weeks previously. Attempts at dilatation were unsuccessful, and on Jan. 31, 1947, a gastrostomy was performed. He was discharged on February 8, and was readmitted on March 12, weighing 114 pounds. He was experiencing difficulty in maintaining adequate nutrition by gastrostomy feedings. On March 20, the cervical esophagus was exposed through an incision on the left side of the neck parallel to the anterior border of the sternocleidomastoid muscle. The strictured area, measuring about one inch in length was incised longitudinally; a nasal tube was passed into the esophagus through the stricture, and into the stomach, where the distal end was brought out through the gastrostomy opening. A string was attached to the lower end of the nasal tube and was pulled through the strictured area and out of the mouth. The tube was then removed and a bougie was placed within the strictured area and anchored by a string attached to each end. The wound was closed in two layers over the bougie. Gentle traction on the string gave early motion to the bougie and prevented the formation of adhesions in the divided strictured area. The wound healed per primum, and the bougie was removed. It was not necessary to continue dilatations during his stay in the hospital. He was discharged on May 2, 1947, with adequate esophageal lumen and had gained in weight from 114 to 145 pounds. He has been followed at intervals and the esophageal lumen continues adequate. When examined on Aug. 20, 1948, he weighed 150 pounds, and stated that he could eat anything he desired.

DR. WILLIAM E. ADAMS, Chicago.—I wish to congratulate Dr. Tuttle and Dr. O'Brien and their group for their demonstration of further advance in this field of surgery. Because of what has been told you by others, I will not go into all the experiments that we have done in this field only to say that our experience supports the thesis that the blood supply of the esophagus is far better than had been anticipated from earlier work. Our experiments might be divided into three groups which I shall mention briefly: One, in which mobilization of the entire thoracic esophagus and division of the left gastric vessels without resection resulted in no deaths from necrosis due to lack of blood supply. A number of these animals died, and it was because of pylorospasm through injury to the vagus. I think that probably explains some of the problems presented here, namely, dilation of the stomach and esophagus. In another group of dogs various lengths up to 6 and 7 cm. of the thoracic esophagus were removed, with primary end-to-end anastomosis. In these animals a liquid diet was given immediately following operation, and no chemotherapy or antibiotics were used. There were no deaths. In the last group of experiments, which are still under way (Dr. Fritz, my associate, is still working on this), the entire thoracic esophagus up to the cervical region is mobilized and brought out through the neck. As much as 6 or 7 cm. of the esophagus is removed and an end-to-end anastomosis made. Of these cases (there are only a few so far), one-third have survived and are getting along well, in spite of the fact that all the blood supply is coming from the left gastric artery.

DR. RICHARD SWEET, Boston.—We have had ample evidence this morning, both experimental and clinical, that localized segments of the esophagus can be resected. I would like to say a few words about what I consider to be the probable limitations of this method as applied to clinical cases, particularly in two diseases. First is the localized stricture which Dr. Tuttle so successfully treated in two of his patients. This is, of course, a relatively rare condition at best and, in my experience, is a somewhat treacherous one from the standpoint of results. Sometimes there seems to be a tendency to recurrence of esophagitis, which is the primary lesion in these cases, just above or close to the anas-

The thoracic and cervical esophagus in two dogs was devascularized without resulting necrosis, but in another animal, operated upon in a similar fashion, gangrene of a portion of the distal segment occurred following transection and anastomosis in the cervical segment.

In two dogs the stomach was delivered into the chest and the esophagus transected 4 inches proximal to the cardia. A portion of the esophagus proximal to the transection was then excised and an anastomosis made to re-establish continuity. Healing was excellent without gangrene.

In four dogs the stomach was delivered into the chest, transections of the esophagus were made 4 to 7 inches proximal to the cardia, and after excising part of the thoracic and cervical esophagus proximal to the transection, anastomoses were made in the neck. In one animal gangrene occurred immediately distal to the suture line.

All intrathoracic procedures were performed through the right pleural cavity.

DR. E. F. PARKER, Charleston, S. C.—We wish also to confirm the experimental observations reported in the first paper. In a series of dogs we were able to resect an average of 33 per cent of the total length of the thoracic esophagus in the dog with a high percentage of success without the use of any adjunctive therapy whatsoever. In the dogs that recovered, some of whom were followed as long as eight months, there was a stricture in only one case. As Dr. Paine mentioned, in several of the other dogs there was dilation of the esophagus proximal to the site of anastomosis without any demonstrable stricture, and we thought this might be due to some neurologic disturbance. We have not had the opportunity to use the operation for benign stricture, to which, of course, it is ideally suited, but we have used it in two cases of carcinoma of the esophagus as a palliative procedure in an attempt to restore swallowing. These were both cases with extension of the carcinoma beyond the point of curability in the field of operation.

The first case was a woman with metastatic carcinoma of the mediastinum and the esophagus, secondary to a carcinoma of the breast removed seven years previously. It was a miserable failure because of disruption of the anastomosis, concomitant invasion of the trachea, and she soon developed an esophagotracheal fistula. In the second case, however, the function of swallowing was restored entirely to normal for a period of three months, at which time the patient developed recurrence of carcinoma at the site of anastomosis. However, the palliative restoration of swallowing definitely made it worth while even though carcinoma did return. Every time an artificial opening is made in one of these patients as a method of presumed palliation, it just adds to their troubles. We are all aware of that but it is well exemplified by a case we had several years ago when a Negro man was treated by the Thorek operation. He got along nicely but of course he had two artificial openings. About six months after operation when he looked very fine, I asked him if he had gone back to work. He said yes, and I asked what he was doing. He said, "It's a twenty-four hour a day job just looking after these two holes."

So if swallowing can be restored in these people without the production of any artificial openings, it is well worth while.

DR. HERBERT MAIER, New York.—I should like to present a case in which resection and end-to-end anastomosis of the esophagus has been utilized as a palliative procedure for a small carcinoma at the junction of the middle and lower third of the esophagus. This patient had metastatic disease which fixed the posterior wall of the stomach to the celiac axis, and it was not possible to free the stomach without transversing this mass. An esophagogastrostomy was therefore not feasible, but by leaving the distal part of the esophagus below the carcinoma attached to the stomach, continuity could be re-established. About 6 or 7 cm. of the esophagus with the small stenotic cancer were removed, and a careful end-to-end esophagoesophagostomy without tension was performed. The operation was performed about one-half a year ago. Roentgenograms show good function of the esophagus with no stenosis at the site of the anastomosis. The patient can swallow well but is now having increasing discomfort due to the intra-abdominal metastases.

it is perhaps worth while to comment on it. The validity of the criticism depends entirely on the question the experiment is designed to answer and the conclusions that are drawn from it.

Dr. Prietto's work is an interesting study of the comparative anatomy of the esophagus of the dog, in which the healing of the tissue is employed as an experimental method. Those of us who keep dogs as pets know full well how they eat, how they bolt their food, and crack and swallow bones. In the dog, the esophagus seems more nearly part of the stomach than it is a specialized conducting tube. The function of the esophagus in the dog, as contrasted with that of man, needs interpretation. Again, the dog has no mediastinum in the sense that the human has. For many years battles were fought on the floor of this Association between the thick pleura animal experimentalists and the thin pleura animal experimentalists. The dog is a thin pleura animal; perhaps he has a "thin pleura esophagus." I hope that Dr. Prietto will not be discouraged. It is only the interpretations that are likely to be drawn from his work that are being challenged by Dr. Gibbon.

DR. PRIETTO.—I wish to thank, on behalf of Dr. Shek and myself, Dr. Churchill and the others who discussed this paper for their kind remarks. Our experiments go only one step at a time; where they will end nobody knows. In addition, may I say, to corroborate the discussions of Dr. Paine and Dr. Parker, I did not mention that in our experiments we did save the vagus, as much as possible without trauma. That might be the explanation, as they concluded, for the dilation of the stomach in one of their dogs which died.

DR. TUTTLE.—I want to thank the discussants. I, too, do not feel that this procedure will ever replace to any great extent the operation of esophageal resection and subsequent esophagogastrostomy. I present this only because it was an interesting observation, because it has worked in these individuals, and because it has not, so far as I know, been previously reported to have been a successful procedure.

In answer to Dr. Sweet, there are two types of lesions which occur in the esophagus which produce strictures and in which there is no previous history as to the true nature of the stricture. We feel that probably one of these things is a peculiar type of esophagitis and I agree that this type of lesion must be resected fairly widely. Two of our patients, I think, had that type of lesion. The second type of lesion was seen in two children. These lesions were probably one of the congenital weblike diaphragms which one sees in children not infrequently. In such lesions, due to the manipulation which is carried out before resection is thought of, there frequently develops a certain amount of esophageal infection, which tends to produce stricture as time goes on.

In answer to Dr. Barnett's comment; I think that probably these are best approached from the left side, because if it becomes apparent that a local resection cannot be done, one might have considerable difficulty from a right-sided approach. Another point is that in the esophageal strictures it is sometimes very hard to tell the exact length by palpation.

In answer to Dr. Gibbon I would like to say that these experiments were done only to demonstrate the blood supply in the dog. Nothing was said about the blood supply in the human esophagus. It was an experimental study on the dog, and the conclusions have been drawn only from the dog. However, it is interesting that healing has occurred, because if one goes back to the older experimental literature it will be found that local resection and end-to-end anastomosis, also in the dog, done fifteen or twenty years ago, almost routinely failed. So I believe these experiments do have a certain amount of merit. We agree with Dr. Gibbon that the studies should be carried further on human beings and in fresh autopsy specimens, and that is our plan. This report was only the first step in a general plan to study the blood supply of the esophagus.

tomotic site. I would venture to predict that, when rather limited segments are removed in this disease, there will be some functional difficulty from recurrence of the same process in certain cases.

The other matter has to do with the localized excision of carcinoma of the esophagus. Of course we all know that this disease is extremely malignant and has a tendency to propagate in the submucosal layers and to extend a long distance beyond the obvious tumor. I doubt, therefore, that this method of local excision will ever have any great application as a means of palliation. I would think that the experience of Dr. Parker, who mentioned that his patient survived for three months before swallowing difficulty recurred, would be the one more apt to be experienced than that of Dr. Maier, whose patient has gone for six months. I doubt whether three months palliation would make it worth while to subject a patient to an operation of such magnitude as this.

DR. JOHN H. GIBBON, JR., Philadelphia.—The blood vascular studies, which have been made in dogs demonstrating the adequacy of the arterial circulation when the short vessels to the esophagus are interrupted, cannot be interpreted as applying to the human esophagus. I agree with Dr. Sweet that probably end-to-end anastomosis of the esophagus will have very little application in humans. I think a study of the blood supply of the human esophagus would be of great value and would apply to those patients in whom an extensive exploration has resulted in interruption of most of the arterial branches to the esophagus in the thorax. I am sure it has been the experience of many of you that you have explored such patients and interrupted a great deal of the blood supply. I have had such experience and fortunately, or perhaps it is the general finding, the patient has not developed gangrene of the esophagus. Arteriograms made in fresh cadavers, studying the anastomotic channels between arterial vessels in the thoracic esophagus, would be of considerable interest. Such injection studies in cadavers, it seems to me, would be more valuable than studies of the esophageal blood supply in different species.

DR. W. EMORY BURNETT, Philadelphia.—Although we are not advocating end-to-end resection of the esophagus for carcinoma, I would like to mention two cases in which Dr. Rosemond, Dr. Cooke, and I did apply that method to carcinoma. I thought it might be worth while to call to your attention these two cases. The first was a pedunculated tumor of 2 to 3 cm. in size on which esophageal biopsy was reported as probably a sarcoma, so that we did approach this from the right side where easy access could be obtained, and did resect locally a distance of about 6 cm. with end-to-end anastomosis. The operative specimen was a pedunculated carcinoma, not sarcoma. That patient has gone more than one year now without symptoms and without evidence of recurrence. The second one was a very small carcinomatous lesion in which we again applied this method of excision and were pleased that the microscopy of the ends of this resected specimen showed no evidence of carcinoma. However, about three months after this the man developed stenosis and of course was thought to have a recurrence. We pushed our luck and again explored this. Because I thought it was not recurrence, we did a very local resection of 2 cm. with frozen section. There was no evidence of carcinoma in this benign stricture. But this patient did not survive the second resection. It was probably poor judgment, to again do end-to-end anastomosis. Those two cases have been our experience with this particular maneuver.

DR. EDWARD D. CHURCHILL, Boston.—Before asking Dr. Prietto to close, I wish to make one general comment because I recall so vividly the bitter resentment that is awakened in a young experimentalist when older surgeons tell him that what he does on the dog is not applicable to the human being. This is because so frequently such a criticism arises from prejudice and ignorance. I would call attention to the fact that Dr. Gibbon is an experienced mammalian experimentalist; yet he made that very familiar remark. If he had not made it I think I would have been inclined to do so, perhaps somewhat more gently. But, knowing the feeling the young surgeon has when such a statement is made,

instrumental dilatation of benign strictures of the esophagus, and severe crushing wounds of the thorax (Table III). There seems to be a correlation between the traumatic agent and the site of communication of the fistula with the respiratory tree. The fistulas caused by compression injury of the thorax communicated with the trachea in 3 out of 4 patients, while those caused by foreign bodies and instrumentation of esophageal strictures communicated with either the right- or left-stem bronchus in 7 of 8 patients.

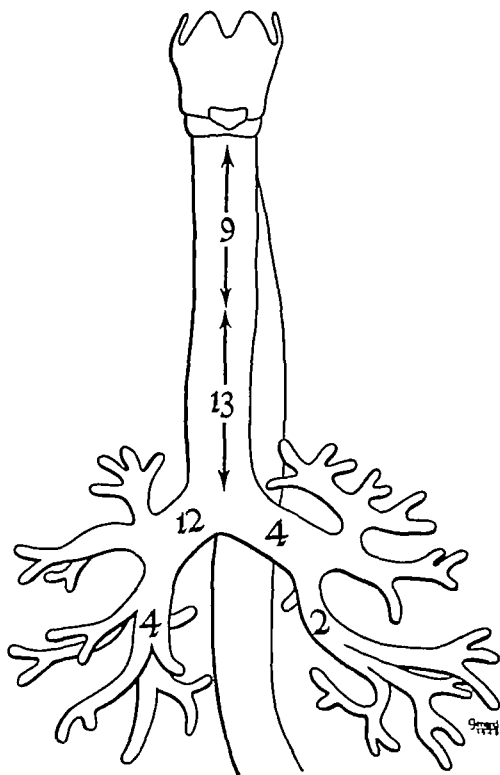


Fig. 1.—Anatomic location of 44 reported cases of esophagotracheobronchial fistula.

Infection was the most common cause of esophagorespiratory fistulas. In a study of the 75 reported cases of nonmalignant fistulas, 26 were known to be caused by a specific type of infection (Table II). Although in 16 additional cases the etiology of the fistula could not be determined by the history, physical

ACQUIRED NONMALIGNANT ESOPHAGOTRACHEOBRONCHIAL FISTULA

FRANK PHILIP COLEMAN, M.D., AND (BY INVITATION) GEORGE H. BUNCH, JR., M.D.
RICHMOND, VA.

THE successful surgical treatment of 4 patients with acquired nonmalignant esophagotracheobronchial fistulas prompted a consideration of the incidence, clinical features, and management of this disease.

We have located in the literature of the past thirty-two years (1916 to 1949) 75 cases of acquired nonmalignant fistulas. Only 6 of these cases were treated by direct surgical repair,^{1, 2, 3, 4, 7, 9} although Poncet¹¹ established the feasibility of this method of treatment in 1896. He successfully closed by curettage and suture through a transtracheal approach in the neck a fistula caused by actinomycosis. The infrequent occurrence of acquired nonmalignant esophagorespiratory fistulas and the paucity of operative cases have led to the accumulation of meager experience by any individual in the management of this disease. We have extracted certain pertinent information from the 75 case reports which we believe will lead to a more comprehensive knowledge of the subject as well as more defined indications for surgical treatment.

The anatomic relationship of the larynx, trachea, and left-stem bronchus to the esophagus is conducive to the development of fistulas. At its commencement the esophagus lies in the midline, but it inclines to the left side as far as the thoracic inlet and gradually passes to the midline again at the level of the bifurcation of the trachea. The esophagus lies in immediate contact with the membranous portion of the trachea and the left-stem bronchus. This relationship may explain previous observations that malignant and nonmalignant esophagorespiratory fistulas more frequently communicate with the left-stem bronchus than the right. Our analysis of the reported nonmalignant acquired variety has yielded 44 case reports in which the location of the fistula was stated (Fig. 1). In this group the right-stem bronchus was involved more often than the left. The deep cervical, paratracheal, and tracheobronchial groups of lymph nodes lie in immediate contact with the esophagus, trachea, and major bronchi, and may play an important role in the pathogenesis of fistulas of infectious origin.

ETIOLOGY

Fifty to 60 per cent of acquired communications between the esophagus and respiratory tract are caused by malignant lesions.^{10, 12} Acquired nonmalignant esophagorespiratory fistulas may best be classified on the basis of etiology (Table I). In an analysis of 75 reported cases, trauma was found to cause the fistula in approximately 1 out of 3 patients (Table II). The most common causes of traumatic fistulas were respiratory or esophageal foreign bodies,

From The Department of Surgery, Medical College of Virginia.

Read at the Twenty-ninth Annual Meeting of The American Association for Thoracic Surgery, New Orleans, La., March 29-31, 1949.

it is not always possible to identify the initial infection. Traction diverticula may lead to fistulous communication with the respiratory tree by continued activity of the initial infection responsible for the diverticulum, by superimposed secondary infection, and by erosion of calcified lymph nodes. The exact location of the opening of the fistula into the respiratory tree was given in 9 of the 11 reported cases of this type. In 7 of these the esophagus communicated with a secondary bronchus, usually the lower lobe, and in 2 the stem bronchus.

CLINICAL FEATURES AND DIAGNOSIS

Symptoms of this disease deserve emphasis. Four cases herein reported were under observation for periods ranging from ten months to eight years without benefit of a correct diagnosis. The development of a communication between the respiratory system and the esophagus invariably leads to characteristic symptoms. The ingestion of fluids is followed by strangling or severe paroxysms of coughing after a momentary asymptomatic pause. In large fistulas solid foods produce the same symptoms and large particles of ingested food may be coughed up. Chronic cough, upper respiratory infections, and repeated attacks of pneumonia are of common occurrence. The fistula may lead to the development of lung abscess or bronchiectasis. Hemoptysis is not an unusual symptom and it may terminate in death.¹⁰ Large fistulas may be associated with severe weight loss. Some patients become accustomed to the inconvenience of the fistula and suffer little interference with nutrition. Many learn the advantage of swallowing in the dorsal recumbent position which tends to minimize the typical paroxysmal attacks of coughing and strangling (Ono's sign).⁸

The diagnosis of esophagorespiratory fistula is based upon the history, physical examination, roentgenographic studies, and endoscopic examinations. Trauma to the chest, ingestion of foreign bodies or caustics, and instrumentation of the esophagus may be followed immediately or later by characteristic symptoms of fistula. Long-standing communications between the esophagus and respiratory tree are usually nonmalignant. Fistulas of short duration in the cancer age group are suggestive of malignant lesions. If dysphagia precedes the development of symptoms of esophagorespiratory communication, it is indicative of cancer of the esophagus. A fistula of tuberculous origin may be preceded by a protracted and indefinite illness (Case 3).

Physical examination may reveal evidence of tuberculosis, syphilis, or mycotic disease. Clinical signs of respiratory tract infection may consist of the presence of moist râles over a lobe or signs of advanced suppurative disease of the lung. Physical signs of pulmonary infection are not always present. A fistula does not invariably lead to chronic suppurative disease of the lung, for the 4 cases herein reported showed no evidence of permanent changes in the tracheobronchial tree. It is possible to demonstrate the fistula in some cases by detecting the appearance of râles in the lungs after the ingestion of water (Ono's sign).⁸ Roentgenographic studies consist of a routine posterior-anterior film of the chest, roentgenograms following the ingestion of contrast media, and bronchograms. Frequently, barium sulfate is administered prior to establishment of the diagnosis; however, ingestion of iodized oil is preferable in that

TABLE I. CLASSIFICATION OF ESOPHAGOTRACHEOBRONCHIAL FISTULAS

I. Congenital
II. Acquired
A. Malignant
B. Nonmalignant
1. Traumatic
a. Foreign body
b. Instrumentation
c. Wounds of thorax
d. Chemical (ingestion caustics)
2. Esophageal diverticulum
3. Infection
a. Tuberculosis
b. Syphilis (gumma or aneurysm)
c. Fungus
d. Nonspecific

findings, and frequently by pathologic examination of excised tissue, it seems likely that many of these resulted from pre-existing mediastinal infection of a nonspecific character. Tuberculosis and syphilis are the most frequent specific infections which lead to fistula formation. Tuberculosis of the esophagus is a rare disease. We have not observed ulcerative tracheobronchial tuberculosis to lead to fistulous communication with the esophagus in the absence of mediastinal tuberculous lymphadenitis. The pathogenesis of tuberculous fistulas is best

TABLE II. ETIOLOGY OF SEVENTY-FIVE REPORTED CASES OF ESOPHAGOTRACHEOBRONCHIAL FISTULA*

Traumatic	21
Undetermined	16
Tuberculosis	14
Diverticulum of esophagus	11
Syphilis	11
Empyema	1
Actinomyces	1

*Includes authors' 4 cases.

explained on the basis of either erosion of caseating or calcified lymph nodes. or rupture of cold abscesses into the tracheobronchial tree and esophagus. Syphilis may involve any part of the respiratory tract or esophagus, and in its tertiary stage may produce fistulas by necrosis and ulceration of gummata. Aneurysms less frequently lead to fistula formation by pressure necrosis.⁶ Two case reports of esophagorespiratory fistula caused by actinomyces appear in the literature.^{11, 13}

Traction diverticulum of the esophagus was responsible for fistula formation in 11 cases. Although traction diverticula can usually be attributed to pre-existing periesophageal infection with subsequent contraction of scar tissue,

TABLE III. ETIOLOGY OF TWENTY-ONE REPORTED CASES OF TRAUMATIC ESOPHAGORESPIRATORY FISTULA

Foreign body	4
Dilatation stricture esophagus	4
Nonpenetrating wound	4
Penetrating wound	2
Operative	2
Chemical (lye)	1
Incomplete data	4

TABLE V. SURGICAL TREATMENT IN TWENTY-TWO REPORTED CASES OF ESOPHAGOTRACHEOBRONCHIAL FISTULA

	NUMBER OF CASES	RESULTS		
		CURED	UNCHANGED	DIED
1. Cauterization (endoscopic)*				
A. Chemical	4	3	1	0
B. Electric	1	0	1	0
Total	5	3	2	0
2. Direct surgical repair†				
A. With preliminary gastrostomy	4	4	0	0
B. Without preliminary gastrostomy†	6	5	0	1
Total	10	9	0	1
3. Pulmonary resection and closure of esophageal defect	2	1	0	1
4. Palliative gastrostomy	3	0	2	1
5. Drainage of coexisting lung abscess	2	2	0	0
Over-all Total	22	15	4	3

*All small fistulas.

†Includes authors' 4 cases.

bronchial tree. It is always necessary to close the defect in the esophagus, whereas the site of communication of the fistula with the respiratory tree may be removed with the resected lobe or lung.

Preoperative preparation of the patient consists of correcting protein depletion, vitamin deficiencies, and anemia, and of restoring the contracted blood volume. Although serious states of nutritional deficiency may exist, we have not found it necessary or desirable to perform a preliminary gastrostomy. Positive nitrogenous balance can be attained in the patient with a large fistula by means of gastric gavage through an indwelling Levine tube. If necessary, this may be supplemented by parenteral nutritional therapy. A survey is made of the cardiac and renal functions. Preoperatively, intramuscular and aerosol penicillin is administered for a period of three or more days. We believe the physical state of the fistulous communication will prove with further experience to be of paramount importance. Smooth epithelized communications are suitable for immediate and direct surgical closure. Small fistulas and fistulas associated with exuberant granulation tissue should receive local cauterization and antibiotics in expectation of cure or alleviation of the local infection preparatory to later surgical treatment.

Caldwell² has directed attention to the problems of anesthesia in acquired communications of the esophagus with the respiratory tree. Large fistulas communicating with the trachea are of little concern to the anesthetist providing there is sufficient space for the insertion of an inflatable balloon between the carina below and the fistulous orifice above (Case 4). If the fistula in the terminal trachea does not permit the interposition of a balloon between it and the carina, a snugly fitting intratracheal tube without a balloon will likely prove satisfactory.² We have had no difficulty with routine intratracheal intubation anesthesia when the fistula communicated with a stem bronchus or secondary bronchus.

it is less irritating to the tracheobronchial tree. Suitable roentgenograms and fluoroscopy following ingestion of opaque oil will usually delineate the course of the fistulous tract. Esophagoscopy reveals the fistula to lie usually in the anterolateral wall of the esophagus. The opening may be easily overlooked due to its slitlike configuration. A beehic blast can be felt when the tip of the esophagoscope is proximal to the orifice of a large fistula. Biopsy is indicated in papillomatous and ulcerative lesions. Bronchoscopy is of value in confirming the communication of the fistulous tract with the respiratory tree. The opening of the fistula lies in the membranous wall of the trachea or bronchus. It may not be possible to visualize the orifice of the fistula in a secondary bronchus; however, it can be localized by injecting methylene blue solution into the esophagus during bronchoscopy. The colored solution may be observed to well up into the involved segmental bronchus. Bronchiectasis must be excluded by adequate bronchograms as its presence alters the surgical treatment of esophago-respiratory fistulas.

TREATMENT

Careful analysis of the 75 reported cases of fistula shows that two-thirds of the patients received no specific treatment. In the group of 53 patients untreated by surgery the outcome was stated in 26; 16 died while hospitalized, 6 showed no improvement, and 4 were cured (Table IV). The results in 22 cases treated by surgery are extremely gratifying. Fifteen of the 22 patients were cured, 3 died, and 4 showed no improvement (Table V).

TABLE IV. NONSURGICAL TREATMENT IN FIFTY-THREE REPORTED CASES OF ESOPHAGO-TRACHEOBRONCHIAL FISTULA

	NUMBER OF CASES	RESULTS			
		NOT STATED	CURED	UNCHANGED	DIED
1. No specific treatment	45	27	1	4	13
2. Antisymphilitic therapy	7	0	2	2	3
3. X-ray therapy	1	0	1	0	0
Total	53	27	4	6	16

The treatment of traumatic esophagorespiratory fistulas requires consideration of the time interval following injury. Acute traumatic fistulas associated with infection or extensive mediastinal emphysema may require external surgical drainage of the mediastinum. Few patients will be seen sufficiently early to warrant immediate operative closure of the fistula because of diagnostic difficulties and the relatively short period of wound contamination.

Fistulas associated with gummatous ulceration of the esophagus or tracheobronchial tree should receive vigorous antisymphilitic therapy. If there is proved or presumptive evidence of tuberculosis in a granulating fistulous tract, recent experience suggests that a preliminary course of streptomycin may be of value. Clerf⁵ has shown that it is possible to close small fistulas (3 mm. or less in diameter) by endoscopic cauterization with silver nitrate or sodium hydroxide. Fistulas complicated by bronchiectasis or lung abscess may require pulmonary resection regardless of the site of communication of the fistula with the tracheo-

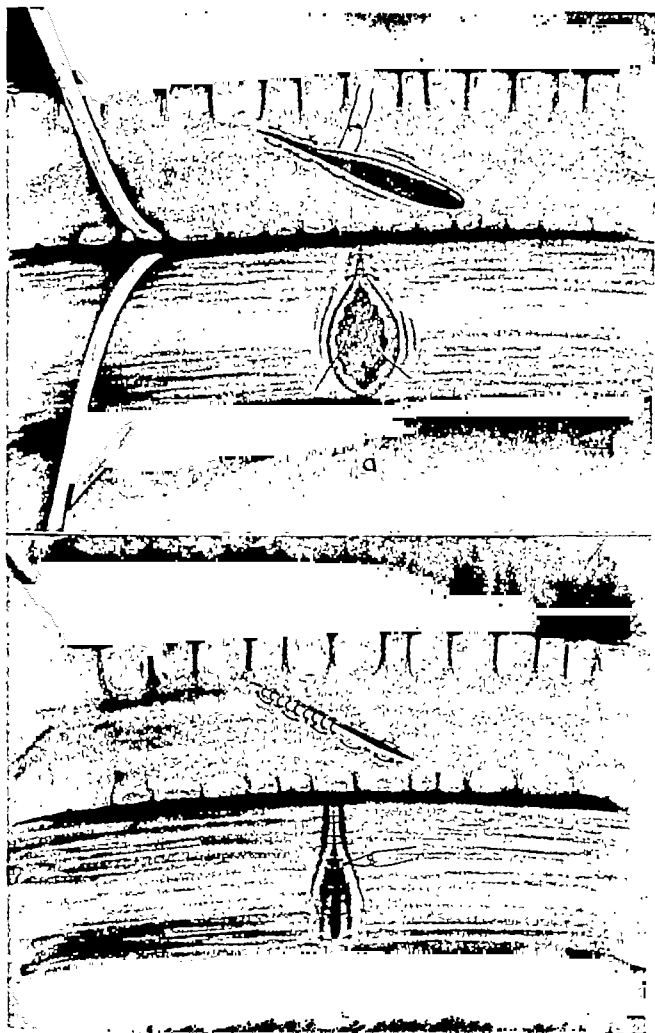


Fig. 2.—Esophageal and tracheal defects following division of fistula (Case 4). *a*, First row of interrupted 000 silk sutures. *b*, Second row of interrupted 000 silk sutures.

Direct surgical repair of an acquired esophagorespiratory fistula requires a cervical, cervical combined with anterior mediastinal, or transpleural approach. Fistulas located in the region of the superior thoracic aperture require a cervical approach combined with either a median sternotomy (Case 4) or resection of the inner third of the clavicle and the anterior portion of the upper two ribs. The esophagus is mobilized above and below the fistulous communication with the respiratory tree. The fistulous tract is divided and if of sufficient length a portion is excised. The defects in the esophagus and trachea or bronchus are closed by one of the conventional methods of suture. We prefer to close the trachea or bronchus with two layers of interrupted 000 silk sutures, a proximal longitudinal mattress suture, and a distal simple on-end suture. The esophageal defect is closed in a transverse plane using two rows of interrupted 000 silk sutures. Simple through and through sutures approximate the walls of the defect and the knots are tied within the lumen of the esophagus. The second layer consists of mattress sutures which approximate the muscular wall over the first layer of sutures (Fig. 2). The repaired defects are protected by the interposition of adjacent tissue.

The postoperative care consists of detailed attention to evacuation of tracheobronchial secretions, removal of the intercostal drainage tube in forty-eight hours (transpleural operation), parenteral penicillin and streptomycin, general supportive measures, and fluids by mouth on the seventh postoperative day.

The following case reports demonstrate the successful surgical treatment of 4 patients with esophagorespiratory fistulas.

CASE REPORTS

CASE 1.—During the early part of November, 1936, E. J., a white woman, 18 years of age, aspirated a small nail into the trachea. An illness followed characterized by severe coughing attacks, high temperature, and production of purulent sputum. A diagnosis of pneumonia was made and after a period of two weeks a bronchoscopist was consulted who failed to find the foreign body. An x-ray film at this time showed the nail to lie in the region of the left-stem bronchus. The fever gradually subsided but she continued to cough, and one month later a second bronchoscopic examination was carried out. This examination showed an ulceration on the posterior wall of the left-stem bronchus. The foreign body was no longer visible in the roentgenogram. Two months after aspiration of the foreign body she began to cough and strangle on the ingestion of liquids. She suffered from three distinct attacks of pneumonia in the lower lobe of the left lung during the ensuing five years. She had been unable to gain weight but complained very little of her symptoms other than strangling and coughing on the ingestion of fluids.

The physical examination revealed an undernourished woman who appeared chronically ill. There was suppression of breath sounds at the left base. The hemoglobin was 72 per cent and the red blood cell count was 3,500,000. The total and differential white blood cell counts were normal. The blood serology was negative. A roentgenogram of the chest showed partial obliteration of the left costophrenic sulcus. Bronchoscopic examination revealed a slitlike defect in the left-stem bronchus 1 cm. below the carina. The fistulous orifice was approximately 1 cm. in diameter in its long axis and there was no associated inflammation. Instillation of colored water into the esophagus at the time of bronchoscopy showed a communication to exist between the esophagus and left-stem bronchus. Esophagoscopy confirmed the presence of a well-epithelized esophagobronchial fistula. After ingestion of Lipiodol

The pathologic examination of the fistulous tract showed it to be well epithelized and lined by squamous epithelium. There was no active inflammation present and no evidence of a specific inflammatory disease.

Intramuscular penicillin was administered for a period of ten days following the operation. On the seventh postoperative day he received fluids by mouth. There were no complications and he was discharged from the hospital on Jan. 22, 1947. He returned for a follow-up examination one month later, at which time he was entirely asymptomatic.

Comment.—A traumatic esophagobronchial fistula 1.5 cm. in diameter in a 46-year-old white man has been apparently cured by resection of the fistulous tract and closure of the esophageal and bronchial defects.

CASE 3.—In November, 1937, E. G., a 63-year-old white woman had what was thought to be a severe case of "influenza" characterized by low-grade fever, ease of fatigue, and loss of weight. This ill-defined state of health persisted for a period of eighteen months. In the early part of 1939 she noticed that ingestion of liquids initiated attacks of coughing and strangling; however, no difficulty was experienced with dry and solid foods. The symptoms showed no marked change until the time of her present hospital admission.

At the age of 43 years she was hospitalized for pulmonary tuberculosis. After a period of six months' observation she was discharged with apparently arrested tuberculosis. As a child she had rheumatic fever unattended by complications. Her father died of pulmonary tuberculosis when she was a child.

This patient was an elderly woman who appeared to be in good health. The physical examination was essentially negative except for moist rales in the lower lobe of the left lung. The hemoglobin was 80 per cent and the red blood cell count was 3,900,000. The total and differential white blood cell counts were normal. The blood serology was negative. The nonprotein nitrogen was 33 mg. per cent. The kidneys were able to concentrate to a maximum specific gravity of 1.015 during the Mosenthal concentration test. The phenol-sulfonphthalein excretory kidney test was normal. The total serum protein was 7.6 Gm. per cent with a normal albumin-globulin ratio. The sputum was repeatedly negative for acid-fast bacilli. The urinalysis showed a one plus albumin with 75 to 100 white blood cells per high powered field. The electrocardiogram revealed abnormal P waves in Leads 2 and 3. The Q₁ and T₁ patterns suggested a residual of old posterior myocardial infarction. The vital capacity was 62 per cent of normal.

The preoperative studies and diagnosis in this patient were made* prior to the patient's hospital admission. A roentgenogram of the chest revealed findings compatible with healed bilateral pulmonary tuberculosis. After the ingestion of a contrast medium, fluoroscopy and suitable roentgenograms demonstrated a fistulous communication of the esophagus with a secondary bronchus of the left lower lobe. The lower lobes of both lungs were filled by the contrast medium and revealed no evidence of bronchial dilatation. The orifice of the fistulous tract could not be identified on bronchoscopic examination. Esophagoscopy showed the orifice of the fistula to be epithelized and to lie in the left anterolateral wall of the esophagus. She was given aerosol and intramuscular penicillin for a period of six days prior to operation.

On Nov. 10, 1947, a left thoracotomy was performed entering the pleural cavity through the periosteal bed of the resected seventh rib. The anesthesia consisted of pentothal and endotracheal gas, oxygen and ether. The upper lobe of the lung was found to be studded with numerous calcified nodules ranging in size from $\frac{1}{2}$ to 1 cm. in diameter. There was partial symphysis of the visceral and parietal pleurae. Multiple calcified lymph nodes were encountered adjacent to the bronchi and in the mediastinum. There was a fistulous tract leading from the esophagus to the posterior segmental bronchus of the left lower lobe 1 cm. in diameter and 4 cm. in length (Fig. 3). The communication was eradicated by excision of 3 cm. of the fistulous tract and closure of the bronchial and esophageal defects as illustrated in Fig. 2.

*By Dr. George A. Weichens, Richmond, Va.

an excellent bronchogram was obtained in that the opaque oil readily entered the left-stem bronchus. The oil filled both the left and right lower lobes and the bronchial pattern was normal.

On Nov. 12, 1941, a left thoracotomy was performed and the esophagus was mobilized above and below its fistulous communication with the left-stem bronchus. A segment of the fistulous tract was excised and the defects in the bronchus and esophagus were both closed with two layers of 000 silk sutures. Sulfadiazine was administered for a period of three days preoperatively and for twelve days postoperatively.

The pathologist reported a tubular structure lined by stratified squamous epithelium.

Convalescence was uneventful and the patient was discharged from the hospital on the twenty-first postoperative day. The patient has remained entirely asymptomatic and there has been no evidence of recurrence of the fistula during the past seven years.

Comment.—A white woman, 18 years of age, with a traumatic esophago-bronchial fistula 1 cm. in diameter and of five years' duration has been cured by resection of the fistulous tract and closure of the esophageal and bronchial defects.

CASE 2.—W. S., a 46-year-old white man, had a nine months' history of paroxysms of coughing and strangling on ingestion of liquids or solids. In February, 1946, he inadvertently swallowed a chicken bone. Seventy-two hours after this incident he noted that the ingestion of liquids or solid food caused episodes of coughing. The symptoms persisted and three months later he coughed up a chicken bone about one-half inch in length. During the ensuing six months he continued to strangle and cough following ingestion of either liquids or solids, and he was unable to maintain a normal state of nutrition. There was a weight loss of thirty pounds.

The physical examination was not remarkable except for evidence of considerable weight loss and severe malnutrition. Rectal examination showed one plus enlargement of the prostate gland. The hemoglobin was 13 Gm. and the red blood cell count was 4,200,000. The total and differential white blood cell counts were normal. The sedimentation rate was 27 mm. the first hour. The blood serology was negative. Roentgenographic study following ingestion of Lipiodol showed the presence of a fistulous communication between the esophagus and left-stem bronchus. A roentgenogram of the chest was normal. Laryngoscopic examination revealed a papilloma of the right vocal cord but both cords exhibited normal function. Bronchoscopy revealed a small patch of granulation tissue on the posterior wall of the left-stem bronchus just below the carina. A dilute solution of methylene blue was instilled into the esophagus with the bronchoscope in place and immediately it was observed to appear in the left-stem bronchus. A diffuse tracheobronchitis was present and associated mucopurulent secretions were aspirated from the left bronchial tree. The communication with the esophagus could not be identified during esophagoscopy.

The preoperative preparation and observation of this patient extended over a period of three weeks. During this time he was repeatedly observed to cough up large particles of food during his meals. The fistulous communication clinically appeared to be much larger than indicated by bronchoscopic examination. It was necessary to supplement the high caloric, high protein diet by parenteral protein hydrolysates and blood transfusions. Intramuscular and aerosol penicillin was administered for a period of one week preoperatively because of associated tracheobronchitis.

On Jan. 3, 1947, a left thoracotomy was performed through the periosteal bed of the resected fifth rib with the patient under pentothal and endotracheal gas, oxygen and ether anesthesia. The esophagus was mobilized above and below its fistulous communication with the left-stem bronchus. The fistula was approximately 3 cm. in length and 1.5 cm. in diameter. The fistulous tract was divided between clamps and the defects in the esophagus and membranous portion of the left-stem bronchus were both closed by two layers of interrupted 000 silk sutures. Bronchoscopy performed immediately after closure of the thorax showed no encroachment on the lumen of the left-stem bronchus.

Over a period of four years he had lost a moderate amount of weight even though he had mastered an ingenious method of preventing food from entering the trachea during deglutition. He had noticed that holding his head slightly to the left and leaning backwards led to improved passage of food into the stomach rather than into the trachea.

There was evidence of moderate weight loss. A few transient râles were heard over the bases of both lungs. The physical examination was otherwise negative. The hemoglobin was 15.2 Gm. and the total and differential white blood cell counts were normal. The sedimentation rate was 21 mm. the first hour. The blood serology was negative. The first and second strength tuberculin skin tests were negative. The vital capacity was normal. The blood chemistry studies were normal. Roentgenographic studies after the ingestion of Lipiodol showed a fistulous communication between the esophagus and trachea at the level of the superior thoracic aperture (Fig. 4). Laryngoscopic examination showed paralysis of the left vocal cord. An ingested silk thread readily entered the trachea by way of the fistula and after being coughed up it was reswallowed. The string made two or three complete revolutions about the described circuit which required its piecemeal removal through a laryngoscope.

Bronchoscopy showed a large fistulous communication of the posterior wall of the trachea with the esophagus at the level of the superior thoracic aperture. The No. 840 bronchoscope could be passed easily into the esophagus by way of the fistulous tract. The fistula had a smooth epithelial lining and appeared to be greater than 3 cm. in diameter. The tracheobronchial tree showed severe and generalized purulent tracheobronchitis. Esophagoscopy showed the fistula to lie in the left anterolateral wall of the esophagus 23 cm. below the upper incisor teeth, and the 9 mm. esophagoscope could be passed through the fistula into the trachea.

The purulent tracheobronchitis required a prolonged preoperative period of aerosol penicillin and streptomycin and intramuscular penicillin. The sputum culture repeatedly showed *Escherichia coli*. It was possible to maintain his nutritional state by a high caloric, high protein diet; however, he never acquired a positive nitrogenous balance. Preoperative transfusions and parenteral protein hydrolyzates supplemented the oral intake.

On Sept. 15, 1948, gas, oxygen, and ether anesthesia were administered through a long endotracheal tube. The attached balloon was placed between the fistula above and the carina below. An incision was made from the level of the superior border of the thyroid cartilage to the suprasternal notch along the medial border of the left sternocleidomastoid muscle. The retropharyngeal space was exposed by retracting the sternocleidomastoid muscle and the carotid sheath structures laterally. The cervical approach was not sufficient for exposure of the fistula and the skin incision was extended downward over the mid-sternum to a level just below the third costal cartilage. The sternum was split in the midline and the anterior mediastinum was opened. The exposure was then satisfactory and the esophagus was freed below the fistula by blunt dissection. The left recurrent laryngeal nerve was observed to be incorporated in a mass of scar tissue in the region of the fistula. The cervical esophagus was developed by blunt dissection above the fistula. There was considerable scarring of the mediastinum in and about the fistulous communication of the esophagus with the trachea. The fistula was divided. The defect in the membranous portion of the trachea measured 5 cm. in its greatest diameter and it was closed with two rows of interrupted 000 silk sutures. A similar defect in the esophagus was closed in a transverse manner using two rows of 000 silk sutures (Fig. 2). The left lobe of the thyroid gland and surrounding scar tissue were sutured over the closed tracheal defect.

Postoperatively, he received 2 Gm. of streptomycin daily and 100,000 units of penicillin intramuscularly every three hours. He developed a wound infection complicated by chondritis of the first, second, and third costal cartilages on the right. On Nov. 1, 1948, the diseased cartilages were resected and the wound was packed open. The wound required secondary closure. Prior to his discharge a bronchoscopy showed no abnormality of the trachea. It

The pathologist reported that representative sections displayed a tubular structure with a central lumen lined by a broad zone of stratified squamous epithelium. The wall exhibited no evidence of an active inflammatory process. No tubercles were present.

Postoperatively, intramuscular penicillin was administered for a period of ten days. She developed atelectasis of the left lower lobe on the second postoperative day which was promptly relieved by intratracheal catheter aspiration. Seventeen days after operation she was discharged from the hospital. She has had no subsequent cough and no recurrence of symptoms during the past fourteen months.

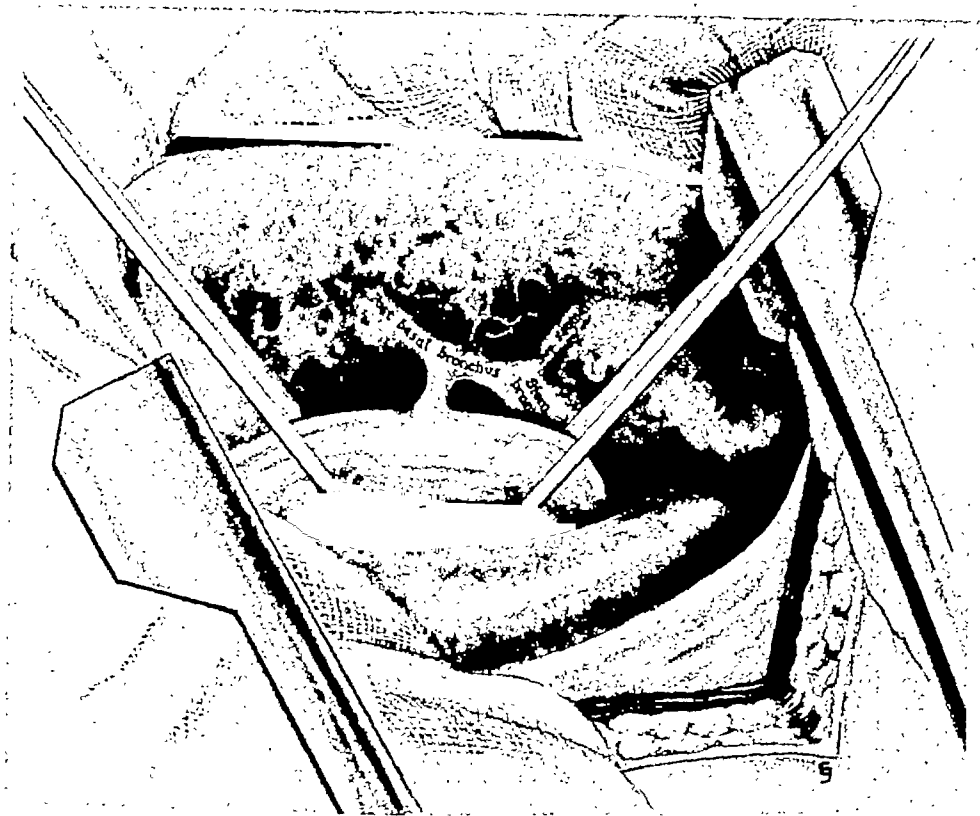


Fig. 3.—Fistulous communication between the esophagus and posterior basal segmental bronchus of lower lobe left lung (Case 3).

Comment.—This 65-year-old white woman had symptoms of an esophago-bronchial fistula for a period of eight years. Coexisting healed pulmonary tuberculosis and calcification of tracheobronchial lymph nodes are presumptive evidence of the fistula's likely being tuberculous in origin, although pathologic examination of the excised fistula showed no tubercles. Excision of the fistulous tract 1 cm. in diameter with closure of the esophageal and bronchial defects resulted in an apparent cure.

CASE 4.—T. M., a 24-year-old white male student, developed "pneumonia" in August, 1944, accompanied by the simultaneous onset of pronounced strangling, choking, and coughing on the ingestion of either liquids or solid food. At the onset of the illness he also became hoarse. He recovered from the "pneumonia" after a period of one week but the associated symptoms persisted to the present hospital admission. He suffered from chronic cough unrelated to swallowing and frequently produced blood-streaked sputum. A second episode of pneumonia was experienced several months after the onset of his symptoms.

SUMMARY

Seventy-five cases of acquired nonmalignant esophagotracheobronchial fistula have been reported in the literature during the past thirty-two years (1916 to 1949). Analysis of these cases has yielded valuable information.

The fistulas are classified on the basis of etiology. The known causes of fistula formation are infection, trauma, and esophageal diverticula. There is a definite correlation between the type of trauma and the location of the fistulous opening in the tracheobronchial tree. Traction diverticula of the esophagus show a high incidence of communication with the secondary bronchi. The most common specific infections leading to fistula formation are tuberculosis and syphilis.

Development of a communication between the respiratory system and esophagus invariably leads to characteristic symptoms. Ingestion of fluids is followed by strangling or severe paroxysms of coughing after a momentary asymptomatic pause. Diagnosis is based upon history, physical examination, roentgenographic studies, and endoscopic examinations.

Fistulas associated with active tuberculosis or syphilis should receive antibiotic therapy. Small fistulas and fistulas associated with exuberant granulation tissue should receive local cauterization and antibiotics in expectation of cure or alleviation of the local infection preparatory to later surgical treatment. Fistulas complicated by bronchiectasis or lung abscess frequently require pulmonary resection in addition to closure of the esophageal defect. Smooth epithelized communications unattended by suppurative disease of the lung are suitable for immediate and direct surgical closure.

Four patients with acquired nonmalignant esophagotracheobronchial fistula have been successfully treated by direct surgical closure. Six patients treated by this method have been reported previously in the literature during the past thirty-two years.

REFERENCES

1. Adams, H. D., and Mabrey, R. E.: Esophagotracheobronchial Fistula Due to Non-penetrating Crushing Injury, *J. THORACIC SURG.* 15: 290, 1946.
2. Berman, J. K., and Walters, C. E.: Empyema Complicated by Broncho-Esophagopulmonary Fistula, *Ann. Surg.* 117: 100, 1943.
3. Caldwell, J. L.: The Anesthetic Management and Problems in Closure of Traumatic Tracheo-Esophageal Fistula in an Adult (Case Report), *Anesthesiology* 7: 317, 1946.
4. Clagett, O. T., Payne, J. H., and Moersch, H. J.: Acquired Esophagotracheobronchial Fistula, *Surg., Gynec. & Obst.* 82: 87, 1946.
5. Clerf, L. H.: Esophagobronchial Fistula; Report of Two Cases, *Tr. Am. Laryng., Rhin. & Otol. Soc.*, pp. 35, 1943.
6. Davidson, P. B., and Mills, E. S.: Fistulae Between Esophagus and Respiratory Tract, *M. Clin. North America* 7: 999, 1923.
7. Judd, D. B., Clagett, O. T., Havens, F. Z., and Moersch, H. J.: Tracheo-Esophageal Fistula; Report of Case, *Proc. Staff Meet., Mayo Clin.* 19: 103, 1944.
8. Lukens, R. M., and Ono, Jo: Syphilitic Tracheoesophageal Fistula; Report of Case, *Laryngoscope* 44: 334, 1934.
9. Moersch, H. J., and Schmidt, H. W.: Esophagobronchial Fistula, *Arch. Otolaryng.* 25: 689, 1937.
10. Moersch, H. J., and Tinney, W. S.: Fistula Between Esophagus and Tracheobronchial Tree, *M. Clin. North America* 28: 1001, 1944.
11. Poncet, M.: Sur un cas de fistule tracheo-oesophagienne d'origine actinomycosique, *Acad. de med., Paris Bull.* 35: 403, 1896.

was impossible to identify the site of the healed surgically closed defect. A laryngoscopic examination showed partial return of function of the left vocal cord. Esophagogram showed no esophageal defects.

At the time of his discharge on Jan. 18, 1949, he had no cough, no difficulty in swallowing, and he had gained twenty-five pounds in weight.



Fig. 4.—Ingestion of Lipiodol delineates the esophagotracheal fistula at the level of the superior thoracic aperture (Case 4).

Comment.—This 24-year-old white man was able to tolerate for a period of four years a fistula which had a diameter of 5 cm. No specific etiologic agent could be demonstrated but there is evidence to suggest that the fistula likely arose from a pre-existing pyogenic mediastinal abscess. The exposure of the fistula at the superior thoracic aperture required a combined cervico-mediastinal approach. Successful closure of the defects in the trachea and esophagus has led to an apparent cure.

alone, and we have had several experiences which have proved that point to us. One in particular was a child in whom a tracheotomy had been performed elsewhere; a large opening had been made in the posterior trachea right into the esophagus. That child was carried along satisfactorily by just utilizing the tracheotomy which was present to keep a good access for secretions, which is one of the factors in conservative management, and the child was fed by means of an indwelling Levine tube. After a period of about sixteen days the fistula was found to be satisfactorily closed. It was of quite long extent, and the child fortunately got no pulmonary complications. A similar incident was in a young Negro boy in whom there was a traumatic wound of large size in the trachea and esophagus, caused by a bullet, and the diagnosis was not made until eighteen hours after its occurrence. Operation had to be done because even two thoracotomy tubes could not control the degree of pneumothorax. Closure was performed, but in view of the amount of contamination the fistula reformed during the postoperative period and closed very rapidly under conservative management with the use of a jejunostomy. I would like to stress the use of esophageal rest, adequate attention to secretions by use of the bronchoscope or tracheotomy tube, and the use of a jejunostomy rather than gastrostomy in these lesions.

DR. JOHN S. HARTER, Louisville, Ky.—I would like to record my experience with three cases of diverticula of the esophagus occurring at about the junction of the lower third and the upper two-thirds. One of these cases was particularly interesting in that we observed the patient for seven years trying to figure out where she was bleeding from. She had frequent hemoptyses and bronchoscopy and esophagoscopy frequently were performed after these hemoptyses, and we never could ascertain where the blood came from. In one of the barium studies an esophageal diverticulum was demonstrated running to the right at about the level of the junction of the lower third and the upper two-thirds. During these seven or eight years of observation she visited various clinics around the country and no one could make a diagnosis. Finally, about 1945, she developed multiple lung abscesses of the lower lobe on the right side and that was resected, and at that time a very definite diverticulum was found extending into the substance of the right lower lobe.

Subsequently two other patients were seen at Nichols General Hospital in Louisville, who were operated on for lung abscess, and diverticula at this level were found extending into the left lower lobe. That is three cases of diverticulum extending into the lobe and because of the inflammatory changes we were never able to demonstrate what the connection was in the lung, except that the diverticulous tract simply ended in the inflammatory tissue.

DR. HERBERT D. ADAMS, Boston.—My experience with this particular lesion has been confined to those of traumatic origin, and I should like to bring up two points, one of which Dr. Abbott has already mentioned, that is, the early management of these lesions. First, it is not necessary to do an emergency procedure. If the patient is in profound shock, either mediastinal drainage or repair of the fistula at that time may be just the thing that will tip the scale and you will lose the patient. At one time, not too long ago, we felt that any rupture of the esophagus was an absolute indication for posterior mediastinotomy and drainage. However, we have proved to our satisfaction, particularly in seriously injured individuals, either with a crushing injury or a penetrating injury which produces an esophago-tracheal fistula, that these patients may be in such severe shock and they may have such extreme difficulty in managing secretions, plus laryngeal edema and emphysema, that it is best to treat the shock, flood them with massive doses of antibiotics, and to do a tracheotomy if necessary to manage the secretions and laryngeal obstruction, and a gastrostomy or jejunostomy to carry on the nutrition and put the area at rest. Having done so, I believe the best approach for these lesions, certainly those below the cervical and superior thoracic strait, is through the right thorax. (Slides.)

The first slide shows a barium swallow, showing how easy it is in some larger fistulas to demonstrate the stream of barium passing from the esophagus into the trachea. The next shows a diagrammatic sketch of the approach through the right thorax. As you know, by dividing the axillary major vein, you will have full access to the esophagus and trachea, and even both the major stem bronchi, and it is possible to mobilize the fistula under direct

12. Sirot, H.: Contribution a l'etude des communications fistuleuses entre l'oesophage et les voies aerinnis, No. 99, Lyon, 1899, 101 pp.
13. Vinson, P. P., and Sutherland, C. G.: Esophagobronchial Fistula Resulting From Actinomycosis, *Radiology* 6: 63, 1926.

SEVENTY-FIVE REPORTED CASES ESOPHAGOTRACHEOBRONCHIAL FISTULA
(1916 TO 1949)

1. Abbott, Osler: *J. THORACIC SURG.* 14: 382, 1945.
2. Adams, H. D., and Mabrey, R. E.: *J. THORACIC SURG.* 15: 290, 1946.
3. Bazy, L., and Chene, P.: *J. de chir.* 45: 370, 1935.
4. Berman, J. K., and Walters, C. E.: *Ann. Surg.* 117: 100, 1943.
5. Blumberger, K.: *Röntgenpraxis* 7: 241, 1935.
6. Butler, C., and del Campo, J. C.: *An. Fac. de med. de Montevideo* 8: 193, 1923. (2 cases.) *Abstract J. A. M. A.* 81: 426, 1923.
7. Caldwell, J. L.: *Anesthesiology* 7: 317, 1946.
8. Chimani, F.: *Wien. klin. Wchnschr.* 56: 150, 1943.
9. Clagett, O. T., Payne, J. H., and Moersch, H. J.: *Surg., Gynec. & Obst.* 82: 87, 1946.
10. Clagett, O. T., and Schmidt, H. W.: *Surgery* 23: 221, 1948.
11. Clerf, L. H.: *Ann. Otol., Rhin. & Laryng.* 42: 920, 1933.
12. Clerf, L. H.: *Tr. Am. Laryng., Rhin. & Otol. Soc.*, pp. 35, 1943. (2 cases.)
13. Coleman, F. P., and Bunch, G. H., Jr.: *J. THORACIC SURG.* 19: 542, 1950. (4 cases.)
14. Girard, L.: *Oto-rhino-laryng. internat.* 20: 294, 1936.
15. Glynn, R. M.: *M. J. Australia* 1: 526, 1933.
16. Grynkrut, B.: *J. de radiol. et d'electrol.* 13: 47, 1929.
17. Gyorgyi, G.: *Röntgenpraxis* 4: 590, 1932.
18. Hawes, J. B.: *Am. J. M. Sc.* 161: 791, 1921. (2 cases.)
19. Kopstein, G., and Schneiderbauer, A.: *Röntgenpraxis* 7: 612, 1935.
20. Krassnigg, M.: *Wien. klin. wchnschr.* 33: 130, 1920.
21. Lebsche, M.: *Deutsche Ztschr. f. Chir.* 220: 249, 1929.
22. Lopes, O.: *Rev. oto-laring. de São Paulo* 4: 1543, 1936.
23. Lukens, R. M., and Ono, Jo: *Laryngoscope* 44: 334, 1934.
24. Mangabeira-Albernaz, P.: *Ann. d'oto-laryng.*, pp. 889, August, 1933. (2 cases.)
25. Melamed, A., and Zimmerman, B. M.: *Am. J. Roentgenol.* 55: 20, 1946.
26. Moersch, H. J., and Tinney, W. S.: *M. Clin. North America* 28: 1001, 1944. (21 cases.)
27. Monserrat, J. L.: *Semana méd.* 2: 760, 1940. (3 cases.)
28. Monserrat, J. L., and Mosquera, J. E.: *Semana méd.* 1: 561, 1942.
29. Murtagh, J. A., and Tyson, M. D.: *New England J. Med.* 222: 494, 1940.
30. Paggi, G.: *Radiol. med.* 20: 1083, 1933.
31. Pape, R.: *Wien. klin. Wchnschr.* 47: 1320, 1934.
32. Paviot, J., Levrat, M., and Guichard, A.: *Arch. d. mal. de l'app. digestif.* 23: 465, 1933.
33. Peco, G.: *Semana méd.* 2: 450, 1929.
34. Piquet, M., Muller, M., and Marchand, M.: *Bronchoscop., esophagoscop. et Gastroscop.*, pp. 27, January, 1939.
35. Pontano, T.: *Policlinico* 35: 348, 1928. (2 cases.)
36. Reveron, V. Marquez: *Arch. venezol. Soc. de oto-rhino-laryng., oftal., neurol.* 3: 206, 1942.
37. Sarazin, M.: *Bull. et mém. Soc. de radiol. méd. de France* 25: 245, 1937.
38. Vinson, P. P.: *Personal communication* (5 cases).
39. Westergren, A., and Ragnell, A.: *Acta Med. Scandinav.* 62: 319, 1925. (2 cases.) *Abstract J. A. M. A.* 85: 1102, 1925.
40. Wohlwool, F.: *Lisboa méd.* 15: 409, 1938.

DISCUSSION

DR. OSLER ABBOTT, Atlanta.—I rise only to mention a factor that I think is important in the handling of these patients; I wonder if any lesion above the level of the aortic arch might possibly have an associated gastrostomy, but certainly below the level of the aortic arch I feel very strongly that if the patient needs additional nutrition he should have a jejunostomy in order to keep the esophagus at rest. Another factor is that a number of such patients may be seen within a period of eighteen to twenty-four or more hours after the acute episode of rupture joining these two organs. In that case immediate operative intervention frequently will be an unhappy factor, and we feel that in that group a particular point should be made in giving them an early jejunostomy, drainage as necessary, and keeping the esophagus at rest, usually by means of Levine suction. A large proportion of immediate traumatic esophagotracheal fistulas will clear under this type of management

THE COMBINED ABDOMINAL AND RIGHT THORACIC APPROACH TO LESIONS OF THE MIDDLE AND UPPER THIRDS OF THE ESOPHAGUS

EDWARD M. KENT, M.D., AND (BY INVITATION) SAMUEL P. HARBISON, M.D.
PITTSBURGH, PA.

IN RECENT years there has been a pronounced increase in attention to both benign and malignant obstructing lesions of the esophagus. This has been due, in large part, to the work of Phemister, Sweet,¹ Garlock,² and others, whose techniques are familiar to all of you. In essence, these principles have meant the adoption of a direct attack upon the problem by the use of esophagectomy and esophagogastrostomy and the abandonment of the indirect approach as demonstrated by the Torek type of operation. These workers have each employed the left transthoracic, transdiaphragmatic approach for lesions at all levels of the esophagus and for those at the cardiac end of the stomach.

In 1946, Ivor Lewis³ published the first account of his two-stage combined abdominal and right thoracic approach for carcinoma of the middle third of the esophagus. At the first stage the abdomen is carefully explored through an upper left paramedian incision. If there are no metastases, the greater curvature of the stomach is freed in its upper half. The vasa brevia, the left gastroepiploic artery, and the omentum are divided well away from the stomach, leaving a vascular arch attached to the greater curvature. The gastrohepatic omentum is then divided well away from the lesser curvature, and the left gastric artery is tied and divided near its origin (Fig. 1). Lewis recommended fine silk throughout for ligatures and emphasized the need for meticulous hemostasis in order to reduce inflammatory changes and the resulting adhesions. This stage is completed by the construction of a Witzel jejunostomy. For a period ranging between seven and fifteen days after the first stage every effort is made to improve the patient's nutrition, and it was the experience of Lewis that the jejunostomy made this possible.

The second stage is performed through the right thorax by removal of the sixth rib and an incision in its bed. If the lesion is found to be resectable, the vena azygos major is divided between ligatures, the involved segment of the gullet is dissected free, the lower esophagus is mobilized, and the portion in the hiatus is freed by digital dissection. The cardia is then drawn up through the hiatus and the peritoneum is divided circumferentially. Adhesions which have formed are gradually liberated as the fundus and body of the stomach are drawn up through the hiatus. When the organ has been sufficiently mobilized to permit placing the fundus well above the level of the cancer, the esophagus is severed at the cardia and the opening in the stomach is closed. The upper end of the stomach is made secure to the mediastinum and chest wall; the esophagus is placed upon the stomach and an end-to-side anastomosis is performed well

Read at the Twenty-ninth Annual Meeting of The American Association for Thoracic Surgery at New Orleans, La., March 29-31, 1949.

and excellent vision, divide the two structures, and close the openings in each with very fine sutures, with an excellent result. These patients have been managed in this manner without residual infection and without pleural contamination.

DR. CAMERON HAIGHT, Ann Arbor.—The paper by Dr. Coleman and Dr. Bunch is quite timely because of the limited recorded experience of any one person in the surgical management of acquired tracheoesophageal fistulas. I would like to refer to one type of fistula which it has been our unfortunate experience to encounter in a few cases of congenital esophageal atresia following primary anastomosis. It concerns those infants in whom there is leakage of the anastomosis with the development of a recurrent tracheoesophageal fistula. This occurs early in the postoperative course and at a time when the tissues are not in a suitable condition for surgical repair of the recurrent fistula. The surgeon is then faced with the alternative of waiting until the condition of the wound improves, or else of trying to close the fistula by chemical cauterization, as advised by Clerf for acquired esophago-bronchial fistulas in adults.

If reoperation is to be done for recurrent tracheoesophageal fistulas in infants, the technical problem is a difficult and uncertain one because of the very considerable scar in the region of the recurrent fistula. Because of these difficulties and also because of the delay which is necessary before reoperation can be done, I now feel that first an attempt should be made to close a recurrent fistula by endoscopic cauterization with a bead of sodium hydroxide fused on the point of a carrier. The cauterization is done under esophagoscopic or bronchoscopic control depending upon which method gives better visualization of the fistula in the particular case. Our last patient with a recurrent fistula was treated successfully by bronchoscopic cauterization, and reoperation was therefore unnecessary.

DR. BUNCH, JR. (closing).—In regard to the remarks of Dr. Abbott, we have not found the need for gastrostomy or jejunostomy in any of our cases. We might point out that neither of these procedures answers the problem of swallowed saliva. Feedings by way of gastrostomy are unsatisfactory in cases with esophagotracheobronchial fistulas, as regurgitation of the feedings is prone to occur through the fistula and into the respiratory tract. As evidence that regurgitation occurs even into the upper esophageal region, we have recently had 3 cases of carcinoma of the cervical esophagus in which the Wookey operation was performed. In each case tube feedings introduced into the stomach escaped rather frequently from the cervical esophagostomy.

We feel that early cases of traumatic fistulas should have drainage of the mediastinum and that nutrition can usually be maintained by Levine tube and intravenous feedings.

Concerning the remarks of Dr. Adams, the posterolateral transpleural approach was used in 3 of our 4 cases, and a combined cervicomedial approach in the fourth. We agree with Dr. Haight that small fistulas should be treated first by local cauterization and the larger ones subjected to direct surgical closure.

The fistulas in our 4 cases were all large. In 2 the etiology was an ingested foreign body, one a small nail and the other a chicken bone. The third case was probably tuberculous in origin as the left upper lobe of the lung was studded with small calcified nodules and many calcified tracheobronchial lymph glands were present. The etiology of our fourth case could not be determined.

The advantages of the right transpleural approach are:

1. Far better access to the upper two-thirds of the thoracic esophagus.
2. Only the azygos vein has to be divided to lay bare the entire esophagus.
3. The aortic arch, instead of an obstacle, becomes a safety barrier between the surgeon and the other pleural cavity. Instead of separating the

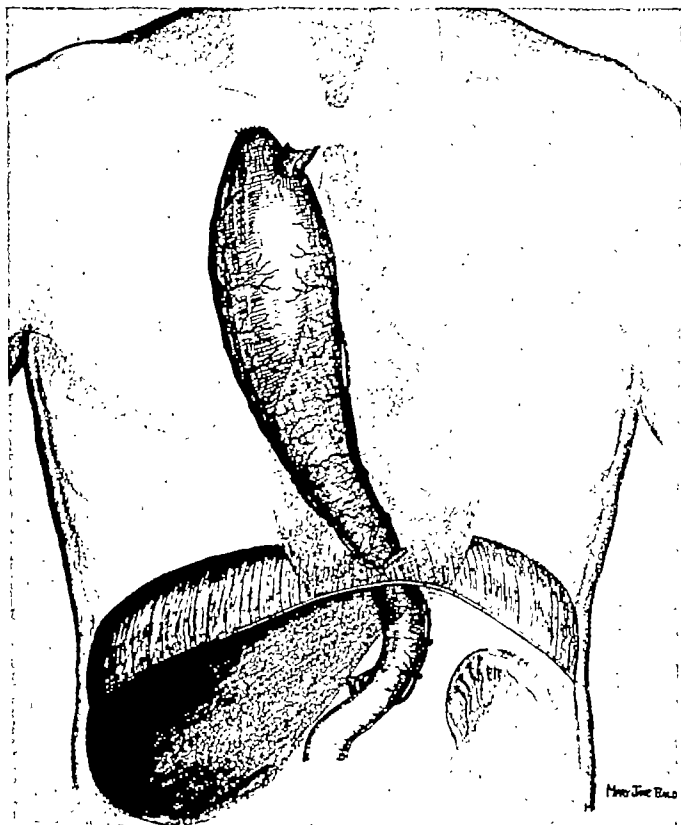


Fig. 2.—Semidiagrammatic illustration of the end result after the right thoracic approach to the esophagus (second stage).

esophagus or its neoplasm bluntly and blindly from behind the arch, it can be dissected under full vision.

In 1948, Macmanus⁴ reported the application of the combined abdominal and right thoracic approach of Lewis in two patients and advocated its use for

above the carcinoma, the esophagus being divided after the first posterior layer of sutures has been completed (Fig. 2). A catheter is placed at the anastomosis line and brought out through an intercostal stabwound to be attached to a water-seal drain. Lewis predicted that both stages could be done at the same time in patients who are exceptional risks for surgery.

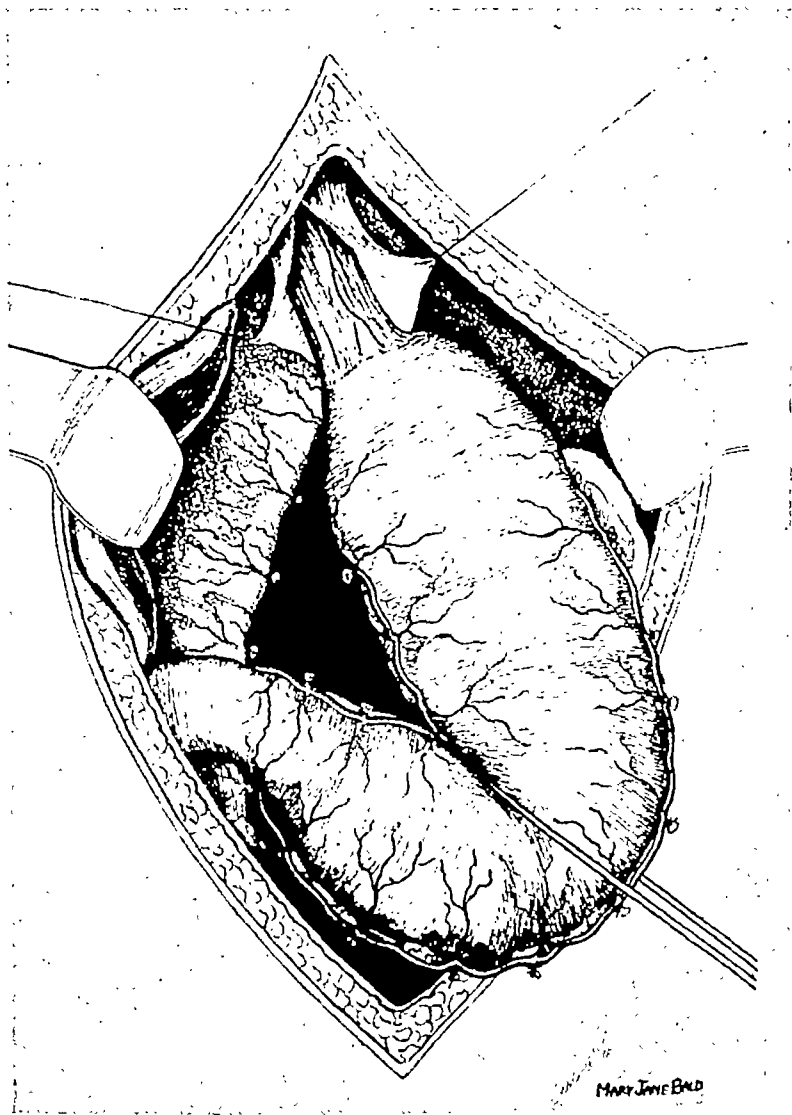


Fig. 1.—Mobilization of the stomach by laparotomy (first stage).

The advantages of the abdominal approach were enumerated by Lewis as follows:

1. A far more satisfactory exploration of the abdomen can be made.
2. A jejunostomy can be performed and is of great value in improving nutrition and hydration between stages and maintaining alimentation in the immediate postoperative period following completion of the second stage.

Since this first success, the Lewis operation has been planned in the surgical treatment of seventeen patients having carcinoma of the middle or upper third of the esophagus. The results are summarized in table form (Table I). As can be seen, all patients were male, only two were less than 60 years of age, all had experienced symptoms for several weeks or longer, and every one had reached the stage of obstruction at which only water would pass, and this with difficulty in some. Only two patients in the group were considered to be good surgical risks. There were five surgical deaths, three after the abdominal stage and two after the thoracic stage. All five deaths occurred within the first twenty-four hours after operation. Four patients received the first stage operation but could not be prepared successfully to withstand the thoracic stage and all four were subsequently released from the hospital. Four patients were found to be not resectable at the thoracic stage, three because of the extent of the disease and one because of cardiac disorders which precluded resection of an operable carcinoma at the level of the azygos vein. All patients in this series had experienced weight loss varying between sixteen and seventy pounds. Several of these patients were found to have metastases in the left gastric nodes at the time of the abdominal stage, but in no such instance was the definitive plan interrupted because of this finding alone. Twelve patients had lesions of the middle third of the esophagus (Fig. 5) while only five were found present in the lower portion of the upper third (Fig. 6). In these latter patients, anastomosis to the upper third of the esophagus was necessary in order that it might be transected well above the lesion.

TABLE I. TABULATION OF FACTORS IN SEVENTEEN PATIENTS HAVING CARCINOMA IN THE MIDDLE AND UPPER THIRDS OF THE ESOPHAGUS

CASE NUMBER	SEX	AGE (YR.)	DURATION OF SYMPTOMS (MO.)	LEVEL OF LESION	RESULT
1	M	52	5	Middle	Successful
2	M	56	4½	Upper	Death after first stage
3	M	60	3	Middle	Successful
4	M	71	4	Middle	Inoperable at second stage
5	M	61	2	Middle	Death after first stage
6	M	68	3	Upper	Death after second stage
7	M	60	2	Middle	Successful
8	M	65	4½	Upper	Inoperable at second stage
9	M	69	2	Middle	Death after first stage
10	M	71	3½	Middle	Inoperable at second stage
11	M	62	4½	Middle	Death after second stage
12	M	67	2	Upper	Successful
13	M	60	1½	Middle	Second stage not undertaken
14	M	69	3½	Middle	Second stage not undertaken
15	M	64	2½	Middle	Second stage not undertaken
16	M	63	5	Upper	Second stage not undertaken
17	M	61	2½	Middle	Inoperable at second stage

It is obvious that in this group of seventeen individuals having esophageal carcinoma the indications for surgery have been extended well beyond the scope of conservatism. This is not typical of the general philosophy of either author for the treatment of carcinoma in most situations. However, we agree with many contributors to the literature on this subject that a rather radical view-

lesions of the lower third as well as for the middle third of the esophagus. The only addition to the technique of Lewis was division of the right crus of the diaphragm at the time the abdominal stage was done.

In August, 1947, we elected to follow the technique of Lewis in the management of an extensive lye stricture which necessitated anastomosis of the stomach to the upper third of the esophagus. The patient had been fed by gastrostomy for twenty-five years and the esophageal lumen had been completely obliterated for many of these years (Fig. 3). Although the result has been extremely satisfactory (Fig. 4), it must be stated that there were difficulties of considerable magnitude along the way. However, none of these were peculiar to this type of surgery but represented hazards which the operator must be prepared to encounter in performing any major surgical procedure. As things



Fig. 3.



Fig. 4.

Fig. 3.—Lye stricture of the esophagus showing dilated lumen above upper limit of the stricture. There was no other lumen in the esophagus except in the lower 8 cm.

Fig. 4.—Roentgenogram showing end result after resection of the esophagus and a right-sided anastomosis of the stomach to the upper esophageal stump.

stand now, this patient is able to eat any foods by mouth although the anastomosis stoma is small and we would like to enlarge it. We have been trying to design a crushing clamp which might be placed on the circumference of the anastomosis in such a fashion as to enlarge the orifice without requiring a thoracotomy. In an instance such as this, the gastrostomy must be closed and this was done at the first or abdominal stage.

Fig. 7.



Fig. 8.

Fig. 7.—Roentgenograms (anteroposterior and lateral) showing carcinoma in the middle third of esophagus. This patient had a posterior gastrojejunostomy which was taken down at the first stage.

Fig. 8.—Anteroposterior and lateral roentgenograms showing a short esophagus, a hiatus hernia, and obstruction at the level of the bifurcation of the trachea. This proved to be adenocarcinoma arising at the cardia of the stomach which was situated in the midthorax.

point is thoroughly warranted. In some instances palliation alone was sought for. Success in this brings rewards of very appreciable magnitude to the patient. On other occasions poor-risk patients have been accepted for surgery in the belief that death from untreated carcinoma of the esophagus, with or without gastrostomy, is a terrible ordeal for both the victim and his family.

As our experience has increased in the management of such cases, it has been our tendency to omit the jejunostomy originally advised by Lewis. This has been based upon our inability to produce good results in improving nutrition thereby. We have also tried to reduce the interval between stages as much as possible, in one instance to twenty-four hours, but in general to a period of from three to seven days, depending, of course, upon the condition of the patient. We



Fig. 5.

Fig. 5.—Roentgenogram demonstrating typical appearance of carcinoma at middle third of esophagus.



Fig. 6.

Fig. 6.—Roentgenogram showing carcinoma in lower portion of upper third of esophagus.

agree with both Lewis and Macmanus that it would be preferable to accomplish both stages at one operation, but in no instance to date has this been feasible. There are two principal reasons for our efforts to reduce the interval to a minimum. First, the longer the second stage is put off, the more adherent the stomach becomes within the abdomen; and second, our inability to accomplish good results with the use of a jejunostomy has been coupled with the observation that the condition of the patient sometimes tends to deteriorate during the interval.

SUMMARY

Experiences in the surgical management of one patient having a lye stricture of the esophagus and seventeen patients presenting carcinoma of the middle or upper thirds of the same structure have been reported. The combined abdominal and right thoracic approach devised by Ivor Lewis has been employed in each instance. Except for the value of jejunostomy, the advantages of this method, cited by Lewis, have been borne out in our own experience. Since successful surgical attack upon such lesions of the esophagus is of recent origin, the methods available to us have not been explored fully as yet. This technique, in our opinion, warrants widespread application so that its place in our armamentarium may be ascertained.

REFERENCES

1. Sweet, R. H.: *J. THORACIC SURG.* 13: 423, 1944.
Idem: *Ann. Surg.* 124: 653, 1946.
2. Garlock, J. H.: *Surg., Gynec. & Obst.* 66: 534, 1938; *Ibid.*: 70: 556, 1940; *Ibid.*: 78: 23, 1944; *Ibid.*: 83: 737, 1946.
3. Lewis, Ivor: *Brit. J. Surg.* 34: 18, 1946.
4. Macmanus, J. E.: *Surgery* 24: 9, 1948.

DISCUSSION

DR. RICHARD SWEET, Boston.—I believe that Dr. Kent's experience presents a very ample demonstration of my opinion that this operation has almost no merit whatsoever. I have long been of the opinion that it would present troublesome difficulties as he has shown and described in his 17 cases. It is almost trite to say that the most obvious objection to it is that, once having put the patient through an extensive abdominal operation for the mobilization of the entire stomach, the surgeon then subjects the patient to another serious operation and finds, as he did in several of his patients, that the disease is hopeless and that the patient must therefore be condemned to go without relief.

He has himself discovered what I learned long ago, that a jejunostomy is of no use or may even present certain objections in the management of these patients. Any patient who is so ill that he appears to require a jejunostomy should not be operated upon. Furthermore, the contention of Mr. Ivor Lewis that the abdominal dissection can be better done through an abdominal incision is wrong. It can be done much more easily and more thoroughly through a thoracic or, as Dr. Garlock has done, through an abdominothoracic incision.

It seems to me that Dr. Kent's mortality is too high, but that may be, as he says, because he has chosen to operate on very advanced cases, and he has a small series. Of course, any small series is apt to be weighted accidentally by unfortunate occurrences. I do not know what his resectability is going to turn out to be when he has done 100 or more of these cases. I might say as a matter of interest that working through the left side (and I realize that it is more difficult technically—still a surgeon must overcome certain technical difficulties—) in my experience at the Massachusetts General Hospital, operating on every patient who was considered to be well enough, it has been possible to resect the tumor in mid-esophageal patients in 72 per cent of cases explored.

If I were to do this combined operation I would do it in one stage; turning the patient over and performing the thoracic portion immediately. As Dr. Kent has learned, interrupting an operation of this sort for a period of days increases the difficulties.

If Dr. Kent or Mr. Lewis can show two things, I will be willing to change my opinion; one is, that his operative mortality is lower than mine; and the other is, that the long time survival of the patients is greater. Until then I shall do it my way.

Two of the patients in this series presented unusual conditions. The first of these, a male patient 52 years of age who had had symptoms for five months, presented an epidermoid carcinoma of the middle third of the esophagus (Fig. 7). The unusual feature in this instance was a high, short loop, posterior gastrojejunostomy which had been performed fourteen years previously for duodenal ulcer. It was necessary to take down this gastrojejunostomy during the abdominal stage.

The second patient presenting an unusual feature was a man 61 years of age. He had experienced dysphagia for two months but had been losing weight for eight months. There was, in addition, pronounced hypertension with impaired cardiac function. An esophagram revealed an obstructing lesion of the middle third of the esophagus at the level of the bifurcation of the trachea with a hiatus hernia (Fig. 8). Esophagoscopy showed the lesion to be well up in the middle third of the esophagus and bronchoscopy demonstrated no evidence of invasion at the carina. The biopsy which was taken at the time of esophagoscopy revealed adenocarcinoma and normal gastric mucosa. We could not understand these observations in tissue obtained at the level of the sixth dorsal vertebra and repeated both the radiographic studies and esophagoscopy with identical results. This is the only patient in the entire group who was found to have adenocarcinoma. The explanation was not revealed until laparotomy was performed and the first stage had been undertaken. A large hiatus was present which would admit the hand, and the cardiac end of the stomach was high in the mediastinum, rather tubular in shape, and conical at the cardia. The esophagus was obviously very short and the cardia lay in the midthorax. The left gastric artery disappeared into the hiatus hernia for some distance before reaching the stomach. The carcinoma had arisen at the cardia and was gastric in origin. Unfortunately, this was one of the patients who did not survive the first stage and again unfortunately, autopsy was refused and this unusual specimen was lost.

We agree that Lewis has made a strong case for his operation and we have found his contentions to have been correct save for our failure to achieve favorable results from jejunostomy. There is no question whatever that access to the esophagus is much better on the right side and that the organ can be mobilized in its entirety with much greater ease than on the left. The abdominal stage has, however, been rather more tedious for us than Lewis has indicated it to be. We have found it quite difficult to mobilize the upper end of the stomach and the lower esophagus in most instances. This part of the operation has been found to be of considerable magnitude as has been shown by the deaths which have occurred thereafter and by our inability to complete both stages at the same time thus far. To our minds, the question rests upon the results of balancing these difficulties against the great advantages of easy accessibility to the esophagus on the right side. We have found the latter to outweigh the former. We are not prepared, however, to agree with Macmanus that this procedure should replace the left thoracic approach for lesions in the lower third of the esophagus.

from least, for a right-handed operator, to anastomose the stomach to a short esophagus in the right chest is much easier than in the left chest. I think Dr. Sweet himself has said that the technical difficulty of making an extremely high anastomosis in the left chest is very great. You will not find that to be true in the right chest.

DR. JOHN H. GIBBON, Jr., Philadelphia.—I have been much interested in this discussion and I must say I agree completely with Dr. Sweet and Dr. Garlock. I take it that one of the reasons for proposing this operation is the difficulty of making an anastomosis high in the chest, that is, a supra-nortic anastomosis on the left side. The mortality in that group is certainly higher than with lesions that lie below the arch, where the anastomosis can be made very easily on the left side. Dr. Sweet and Dr. Garlock have both reported cervical esophagogastronomies; Dr. Garlock's patients unfortunately died early, but Dr. Sweet's patient lived. Dr. Lyman Brewer of Los Angeles has recently reported such a case, and so has Dr. Wylie of New York. Dr. Frank Allbritten and myself have recently had 3 cases of cervical esophagogastronomy. One of these patients died, the other 2 are living and well at present.

The anastomosis is much easier in the neck than it is high in the chest. One of our successful cases was a complete esophagectomy. The man is still living five months later, in good health except that he has a carcinoma of the rectum which he refuses to have operated upon. It was a total esophagectomy, anastomosing the stomach to the pharynx. I think the feasibility of these cervical anastomoses for lesions in the upper third of the esophagus does away with the arguments advanced for the approach from the right side of the chest.

DR. J. M. CHAMBERLAIN, New York.—It takes a brave man to stand up at this time and defend Dr. Kent, especially after Dr. Garlock and Dr. Sweet's opening discussion. I imagine that it is because of my comparatively small experience that I have the courage to do so. I just want to mention two small points. I should like to emphasize the importance of preserving the intact diaphragm to which Dr. Paine has already referred. Many of these patients, as you all know, have a very low respiratory reserve, either because of emphysema or later because of some postoperative complication, and a good technical result may be a clinical failure simply because of respiratory insufficiency. Bronchial peristalsis and the cough mechanism are Nature's protection against these postoperative complications, and the diaphragm of course is extremely important in both. If the diaphragm is crippled by Dr. Sweet's operation, it is conceivable that some of these patients are lost simply because of pulmonary complications, as has happened in one of our cases. We have used fluoroscopy on many of our patients on the first postoperative day, and the lateral half of the diaphragm does not move and the medial half moves but slightly, so there is no doubt that the diaphragm is crippled as the result of the thoracoabdominal approach.

My second point is in defense of the right-sided approach and has to do with the resectability of the high lesion. For a long time I avoided the right-sided approach because I disliked putting the patient through a major abdominal stage only to find, upon opening the chest, that the tumor could not be resected. It is a matter of mental gymnastics, actually, to change a whole philosophy and assume that they are all operable or the obstruction can be shunted. Dr. Pool got around this by anastomosing the stomach above the lesion regardless of whether or not the tumor is removable. So in our last several cases we have assumed that all cases are resectable, not 33 out of 60, which is about 50 per cent, and not 72 out of 100—but all are resectable in the sense that if the stomach can be mobilized an anastomosis may be accomplished above the lesion. As much of the lesion as possible is then removed. We do both stages immediately. The stomach is brought up through the hiatus, without opening the crux; and the "shunt" is done regardless of whether all the cancer is removed or not. The older surgeons here probably have nothing but contempt for the gastrostomy procedure but the shunting operation, at least in our small experience, has led us to believe that the patient lives three to six months more happily than he would with a gastrostomy.

DR. JOHN H. GARLOCK, New York.—I feel exactly as Dr. Sweet does, although I do not think I could put it as succinctly as he did. While I was in London in 1947, I talked to Mr. Lewis about this operation and he indicated that the great difficulty in this right-sided approach with the preliminary abdominal exploration and mobilization of the stomach, resulted frequently in the finding at the second stage of a hopelessly inoperable lesion which could not even be given the benefit of palliative resection. My main objections are two; the first I have already indicated; the second is that you face the patient with a real operative risk in the mobilization operation done at the first stage. Instead of accomplishing the operation in one stage, you give the patient two risks of major proportions.

DR. JOHN POOL, New York.—I would like to report an experience of six cases in which this procedure was done, in four of whom the lesion was resectable and in two of whom, where the lesion was not resectable, a by-pass procedure was carried out so that the patient was left with a continuity of digestive tract, and the operation was not, therefore, without benefit to the patient. (Slides.)

I have here, just for comparison, two patients in whom the left approach for the lower quarter of the esophagus, approach through the left side of the thorax, did not allow resection of the esophageal carcinoma, but by simple mobilization of the fundus of the stomach through the diaphragm a by-pass anastomosis could be effected at this point and continuity restored. You can see some reflux of barium in the distal esophagus at this point. Here also is another demonstration of such a by-pass, with again reflux of barium in the inferior portion of the esophagus. I do not believe that this procedure has materially prolonged life in these patients; they both survived approximately six months, but during that time they were able to swallow and there was not recurrence to the point of blockage of the carcinoma at the level of the anastomosis although it was quite close to the upper limit of the disease. This slide will show you a very extensive carcinoma in which the Ivor Lewis procedure was carried out. It was not possible on exploring the chest to mobilize the carcinoma, but the stomach could be brought up and an anastomosis performed above the level of the carcinoma. This was done in one stage.

DR. J. R. PAINE, Buffalo.—I am sorry my colleague Dr. Macmanus is not here to discuss this paper. I also hope that Dr. Sweet and I do not cease to be friends. I also hope that he has tried a right side approach when he speaks as he has. I had the same impression Dr. Sweet had, largely because I followed his writings on the subject, until I tried it. Dr. McManus convinced me that it was a worth-while procedure. We have pooled our efforts, and I would like to show you our results.

We have operated upon 60 patients with carcinoma of the esophagus. We have been able to resect 33. There have been 7 deaths in the total number. We have used the left abdominal right thoracic approach in 27 patients, and of the 27 we have been able to resect in 17. Only 2 of the 27 have been done in two stages; 25 have been done at one stage. That makes a rather long operation but the patients have stood it, I think, as well as they would any type of operation. There are very definite disadvantages and very definite advantages to this operation. The two major disadvantages have already been mentioned. A major surgical procedure is performed before resectability is determined, but we sometimes do major surgical procedures before we determine the resectability of many tumors. An adequate closure of the hiatus about the stomach is very difficult; I doubt if that is a disadvantage. In the majority of our cases we have made no attempt to suture the hiatus to the pyloric portion of the stomach after it has been brought into the right chest.

There are definite advantages, too; a very adequate amount of proximal esophagus may be removed without increasing the difficulty or mortality of the operation. Both phrenic nerves and diaphragm remain intact, and that, we think, is a very important point. The respiratory mechanism is left as complete as possible after operation. The thorax is opened for a shorter time, thus disturbing the cardiorespiratory mechanics for a much shorter period than when using the left approach. The incidence, in our experience at least, of post-operative atelectasis and pneumonitis has been less. Interference by the aortic arch is less. In only 3 instances have we had any trouble with cardiac irregularity. And last, but far

tube was then readily available in the pneumothorax space at the second stage and made the technique simplified. This might be helpful if a two-stage, right-sided approach as reported is to be employed.

However, as the various advances in esophageal surgery developed it was found that high anastomoses could be accomplished without the use of reversed gastric tubes. The first total thoracic esophagectomies and esophagogastric anastomoses performed by me were also accomplished by utilizing two incisions, first an abdominal one and then a thoracic one, done in one stage. The use of two incisions was also found to be time consuming and unnecessary. Adequate exposure for both the thoracic and abdominal aspects of the operation can be obtained through the thoracic incision. The exposure is through the bed of the sixth rib. Occasionally additional exposure for the abdominal aspect is obtained by resecting the seventh rib anteriorly and moving the self-retaining retractor forward. It is also possible to resect the fifth rib posteriorly to allow better exposure for the anastomosis.

Thirteen total thoracic esophagectomies and anastomoses performed at the apex of the chest as well as one performed at the level of the pharynx have been accomplished in this manner with what was felt to be adequate exposure.

DR. W. EMORY BURNETT, Philadelphia.—I should like to bring to your attention something which I am sure many of you have tried in the exposure of the high anastomosis on the left. After doing the mobilization through the seventh, eighth, or ninth ribs as usual, then finding it difficult to reach the high level, we have, on two occasions made another incision through the resected third rib bed and found it made the anastomosis considerably easier; it apparently did not increase the risk although it might be preferable to have a fairly good risk patient for extending the operation in that way. However, it shortens the time of anastomosis and this compensates for the extra incision.

DR. HARBISON.—It is presumptuous to make any further comment on this subject. I realize it is highly controversial and difficult to evaluate. I feel very strongly that Dr. Sweet should try some of these because I would like to see what his technical skill would accomplish in that operation and to have some comments from that side. Certainly the only real advantages that I can see are the better resectability of the lesion from the right with the aortic arch out of the way, better resectability from the viewpoint of cancer surgery. The one-stage operation is obviously the best, and we have attempted it, but it has been a question of poor-risk patients who were not able to stand it.

DR. KENT.—There is no reason for me to speak except that I must acknowledge and share with Dr. Harbison the criticism which has been directed at this presentation. We are fully aware that the results shown here today provide excellent ammunition for critical attack, nevertheless we persist in our opinion that these results are due primarily to the poor selection of patients for surgery, or rather, to the acceptance of poor-risk patients for operation.

We expected sharp criticism, we welcomed it, and we would have been disappointed had it not been forthcoming. We believe that in recent years the meetings of this Association have changed in many respects and that one of the more serious differences has been the constantly agreeable character of our discussion from the floor. We are happy to have been responsible for a revival of such spirited discussion.

DR. JULIAN JOHNSON, Philadelphia.—If one desires to perform an esophageal resection from the right-sided approach, from choice or necessity, there would seem to be little point in doing it in two stages. We open the right chest first to demonstrate that the lesion is resectable. The patient may then be turned back slightly and a left subcostal incision made to partially free the stomach from its vascular attachments. An assistant working on the abdomen may then pass the stomach up to the operator working on the thorax. This plan has worked out quite satisfactorily for us and has the advantage of determining operability early.

DR. CHARLES P. BAILEY, Wyncote, Pa.—We have usually employed the left-sided approach for esophageal resection at all levels, but recently, following experimental work by Dr. Victor Satinsky, who is associated with us, we have attempted a one-stage, right-sided thoracoabdominal approach for high esophageal lesions.

In addition to wide severance of the diaphragm, and mobilization of the esophagus and stomach from the right side, our technique includes division of the esophagus at the gastroesophageal juncture, closure of the esophageal hiatus, and introduction of the stomach into the chest through a new hiatus in the right posterior diaphragm. Since the stomach now ascends into the thorax in a straight line, directly from the pylorus, greater length is afforded for high anastomosis.

This approach was very effective in dogs, but unfortunately did not end happily in 2 cases tried so far. The first was a poor risk patient who had a huge tumor occupying about two-thirds of the esophagus, and at the time of operation it was adherent to the arch of the aorta in such a way that I believe it could not have been removed had we gone in on the left side. I must admit that after it was over I wondered whether it would not be a good idea to go in on the left side in such a case because then you would not be tempted to take it out. However, we did succeed in extirpating the tumor and establishing an esophagogastrostomy with ease. The patient did well for twenty-four hours, then suddenly died, apparently of a cardiac complication; he was a very seriously ill man and had had an obstruction for some weeks prior to surgery.

The second case was a man in rather good condition who had a high esophageal lesion. Anastomosis was established by means of our one-stage, right-sided thoracoabdominal approach, and the patient was in good condition at the end of the procedure. The next day, however, he turned quite yellow and showed all the signs of uncontrollable hemolytic blood transfusion reaction, and died after six days from that cause. At autopsy we found an excellent primary healing of the anastomosis, and everything about the operation seemed to be good.

I thought it best to report these 2 cases because no one seems to have performed quite such a procedure. I hope we will have better success with our approach in the future. Meanwhile, a detailed account of the technique is being prepared for publication.

DR. EARLE B. KAY, Cleveland.—To solve the problem of high esophagogastric anastomosis, I spent some time in the experimental laboratory in 1941, in an attempt to find a technique applicable to high esophagogastric anastomosis.

The first method employed was the use of reversed gastric tubes. The operation was divided into two stages. The first stage consisted of fashioning a reversed Beck-Jianu gastric tube through an abdominal incision. The second stage performed seven to fourteen days later consisted of resecting the esophagus. The previously prepared gastric tube was then brought through the diaphragm for the anastomosis.

The second stage was always very difficult because of the development of adhesions about the previously mobilized stomach and gastric tube. In one patient upon whom this procedure was attempted, adhesions prevented adequate mobilization of the gastric tube. The increased technical difficulty caused by adhesions is a very definite criticism of any two-staged procedure as described in this paper.

To obviate this difficulty the operation was then changed so that the gastric tube was inserted through the diaphragm into a pneumothorax space within the chest. The gastric

Kegaries' observations may very well be repeated at this time: "It is probable that the capillary-like anastomosis at the cardia offers a marked resistance to the flow of blood from the portal to the systemic veins as in the case when the portal circulation becomes obstructed. In such instances the increase of venous pressure must exert itself upon the veins below the cardia for some time before the resistance of the cardia is overcome. Varices result from an increased flow of blood through a vein and an inability of the muscular and elastic fibers to withstand an increased intravenous pressure at the cardia; and as has been noted previously, the submucous veins of the esophagus are well supported and it is reasonable to believe that the formation of varices first occurs below the cardia and secondly above that point after collateral channels of the anastomosis have been established. *Where several veins of large caliber cross the cardia, the formation of varices above the cardia is probably more rapid. When present they are no doubt the first veins in the region of the cardia to become enlarged.*"

There is no need in this paper to enter into any extended discussion concerning the causative factors in the production of portal hypertension. Whether the cause is intrahepatic (cirrhosis of the liver) or extrahepatic (thromboses of the splenic and/or portal veins, cavernomatous transformation of the portal vein, external portal vein compression, etc.) the effect is the same and is measured in terms of a markedly increased pressure within the portal venous system. The finding of venous dilatations, whether they be located at the cardia and lower esophagus, in the hemorrhoidal area, along the falciform ligament of the liver or the retroperitoneal area, is evidence of an attempt at establishing a compensatory mechanism, the main purpose of which is to reduce the pressure in the greater splanchnic area and portal bed by shunting blood into the caval system. All observers of the general symptom-complex of portal hypertension are agreed that the most alarming symptom, one prone to cause death of the patient quickly, is bleeding from esophageal varices. Therefore, all therapeutic efforts have been directed towards eliminating or greatly minimizing this all too frequently fatal complication.

Since 1894, when Banti first suggested splenectomy, many remedies have been suggested and tried. These included: (1) omentopexy; (2) portocaval anastomosis, first successfully consummated by Vidal in 1903; (3) various vein anastomoses; (4) ligation of various gastric vessels; (5) injection of sclerosing solutions into esophageal varices, first performed by Crafoord and later popularized by Moersch; (6) suture and nonsuture anastomoses between portal vein and inferior vena cava and splenic to renal veins; (7) extensive gastric resection (Wangensteen); and (8) total gastrectomy (Phemister and Humphreys).

Most of these measures have long since been abandoned because they were found to be ineffectual in controlling the bleeding. The portocaval shunt operation and the splenorenal vein anastomosis, advocated by Blakemore and Whipple, have more physiologic basis than any procedure heretofore suggested. Yet, even these more logical operations are time consuming and carry with them

FURTHER OBSERVATIONS ON PACKING OF MEDIASTINUM FOR ESOPHAGEAL VARICES

JOHN H. GARLOCK, M.D., AND (BY INVITATION) MAX L. SOM, M.D.
NEW YORK, N. Y.

IN NOVEMBER, 1947, we reported our experience with two cases of severe bleeding due to varices of the esophagus treated by gauze packing of the upper thoracic mediastinum. The purpose of this procedure was the formation of granulation tissue in the mediastinum surrounding the upper thoracic esophagus in the hope that there would develop in this tissue an extensive periesophageal venous plexus capable of taking the load from and reducing the pressure in the varices themselves, thus shunting more blood from the portal to the caval circulation. Experiences with six additional patients, eight in all, form the basis of this report.

It is important to stress the pathologic anatomy of the venous blockade in portal hypertension, especially in the light of the observations made by Baronofsky in a recent article published in *Surgery*. It seems to us that the most important area of contention centers about the cardiac end of the stomach and lower esophagus. It is well known that, except in rare instances, the lower esophageal veins drain into the caval system and the veins in the upper stomach empty into the portal system, mainly through the left gastric vein. That there also exists a fine anastomotic connection between the lower esophageal and cardiac veins, thus uniting the portal and caval circulations at this point, there can be no doubt. The injection studies of Kegaries, in 1934, definitely established this. These anastomotic channels lie in both the submucosal and submuscular areas, as do also the regularly demonstrated veins in the lower esophagus and upper stomach.

It is also well known that not every case of portal hypertension, whether of intrahepatic or extrahepatic origin, is complicated by bleeding from esophageal varices. It seems clear to us, therefore, that the reason for this is to be found in the extent and degree of the anastomotic network between the caval and portal systems at the cardiac and lower esophageal areas. With a poorly developed communicating network, the chances for compensatory dilatation of the lower esophageal veins would be greatly minimized. In the presence of a rich anastomosis, the increasing pressure in the portal system would be transmitted more easily through these communicating channels into the esophageal venous system, thus causing the formation of submucosal varices. In the presence of a poorly developed communicating system, the compensatory mechanism in this area takes other directions, mainly through the azygos and diaphragmatic veins. However, it is well known that these latter communications may also be poorly developed.

From the Surgical Service and the Laryngological Service of Dr. Rudolph Kramer, Mt. Sinai Hospital.

Read at the Twenty-ninth Annual Meeting of The American Association for Thoracic Surgery, New Orleans, La., March 29-31, 1949.

We have approached the problem of bleeding from esophageal varices in a more direct way in that advantage is taken of the communicating system at the cardia between the portal and caval circulations by attempting to reroute the portal blood from the vulnerable submucosal varices to a surgically produced new periesophageal venous plexus. The beneficial effect in the first case was obtained quite by accident. This patient sustained a perforation of the cervical esophagus during esophagoscopy for varices following a severe hepatitis a year before. The infection of the posterosuperior mediastinum was drained by another surgeon at the hospital through a cervical incision and packing was inserted. The subsequent complete abatement of hematemesis in this patient led to the use of this procedure as a definitive measure in the treatment of bleeding from esophageal varices secondary to portal hypertension.

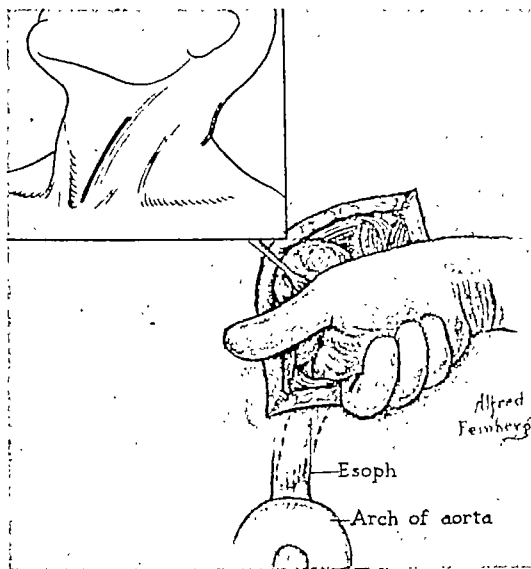


Fig. 1.—Upper mediastinal packing through cervical incision. Blunt separation of posterior and lateral surfaces of esophagus from prevertebral fascia almost to upper surface of arch of aorta.

Since our first report in which it was suggested that packing of the posterosuperior mediastinum should be tried, we have extended the operation to include, as a second stage in selected cases, packing of the entire posterior mediastinum from the arch of the aorta to the diaphragm through a right transpleural approach. Two of our patients have been treated by the latter method.

The first procedure offers no difficulties in its performance. Through a left oblique cervical incision along the anterior border of the sternomastoid muscle, the retroesophageal area is exposed by retracting the carotid sheath

a high operative mortality. It is possible that further experience will indicate more clearly which patients should or should not be accorded these operations and that eventually the operative mortality will fall to a more reasonable level.

The observations of Wangensteen and Baronofsky are provocative and open to considerable discussion. On their assumption that it is unreasonable to believe that mere hydrostatic distension of the esophageal varices and their eventual rupture is the sole cause of the hematemesis, they advance the theory that, because of impairment of nutrition of the mucous membrane overlying the varices due to venous pooling, these tissues present a lowered resistance to the action of gastric digestive juices. The initial factor, therefore, becomes a chemical destruction of the impaired mucous membrane, followed subsequently by rupture of the unprotected varices. They, therefore, recommend almost total gastrectomy for three reasons: (1) to remove the acid peptic factor; (2) if there is a communication between the portal and caval systems at the cardia, then gastrectomy should isolate the esophagus from the portal system; (3) gastrectomy reduces the blood inflow into the portal circulation.

There comes to mind immediately a number of sound reasons why this theory is untenable. In the first place, on the basis of innumerable direct observations of the esophagus through the esophagoscope, one of us (M. L. S.) has never seen mucous membrane erosions in this disease which could be interpreted as being caused by regurgitation of gastric juice. The latter has been observed frequently in peptic esophagitis and benign peptic ulcer of the lower esophagus. Second, those of us who have had any considerable experience with surgery of the esophagus are well acquainted with the frequent extreme friability and remarkable thinness of the mucous membrane of this organ. It is not difficult to imagine a markedly distended vein beneath such mucous membrane giving way under the repeated trauma of swallowing large or hard boluses of food. This we believe to be the logical mechanism of the bleeding in this condition. Third, it is difficult for us to understand why a high gastric resection or a total gastrectomy should separate the portal from the caval circulations. The jejunum, which is utilized in effecting these anastomoses, is as much a part of the portal backflow as is the stomach. There is every reason to suppose, therefore, that following the gastrectomy, anastomoses will be effected between the jejunum and the upper stomach or esophagus, thus re-establishing the connection between the portal and caval systems. Inasmuch as most of the esophageal veins are located in the submucosal area, the back pressure will be felt again in the varices and hematemesis should recur.

Of the eight patients reported by Baronofsky from Wangensteen's Clinic, four died postoperatively. The case descriptions indicate that the latter were very poor operative risks. Of the four survivors, one died nine months after a 90 per cent gastric resection following a severe hematemesis. The second patient, a boy 3 years of age, had one bleeding episode two years and five months after a 98 per cent gastric resection. The third has had no further bleeding for two years following a 95 per cent resection. The fourth patient had a total gastrectomy and has remained symptom free for seven months.

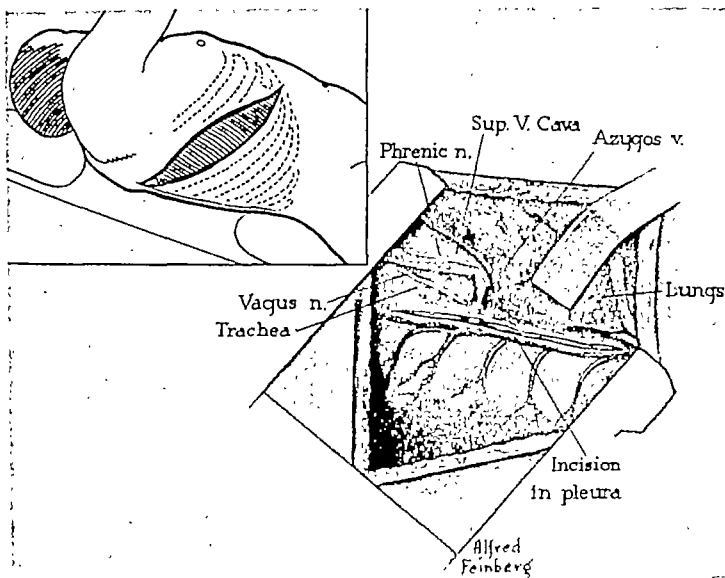


Fig. 4.—Packing of posterior mediastinum. Right transpleural approach with or without resection of seventh rib. Long incision in mediastinal pleura overlying esophagus. Fashioning of two flaps of pleura to be used for final closure.

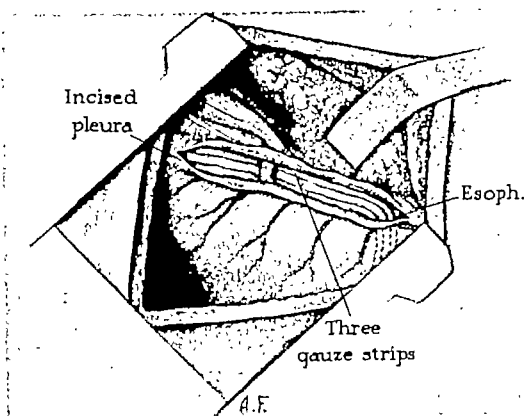


Fig. 5.—Three strips of one inch single thickness gauze packing are placed over esophagus in longitudinal direction. The emerging ends are drawn beneath lateral pleural flap in a channel created between parietal pleura and rib cage to emerge from chest wall in a sub-jacent intercostal space.

laterally and the thyroid lobe mesially. The lateral and posterior surfaces of the esophagus are bluntly separated from the prevertebral fascia as far down in the mediastinum as the finger will reach, usually in the neighborhood of the arch of the aorta. Into this space is packed dry plain gauze, not too tightly,

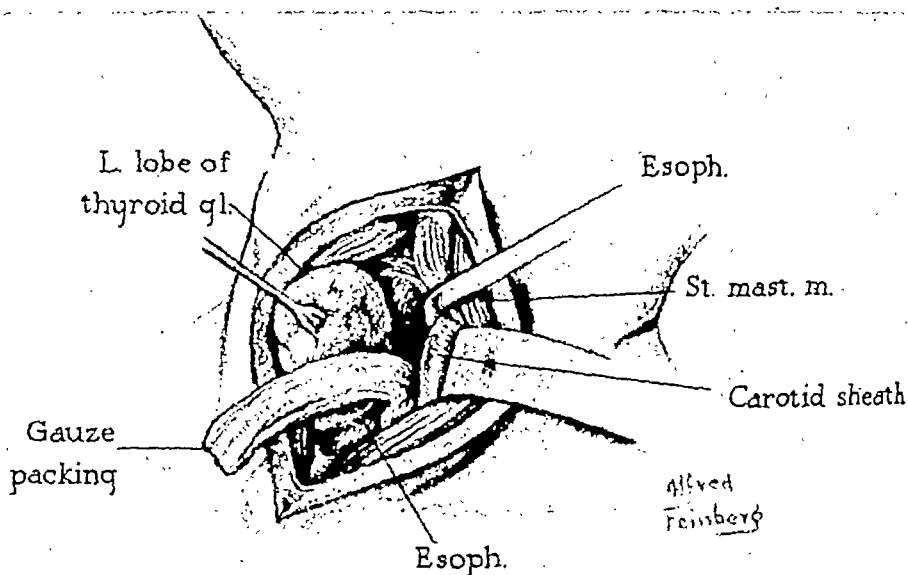


Fig. 2.—Insertion of plain gauze packing into space created by blunt dissection.

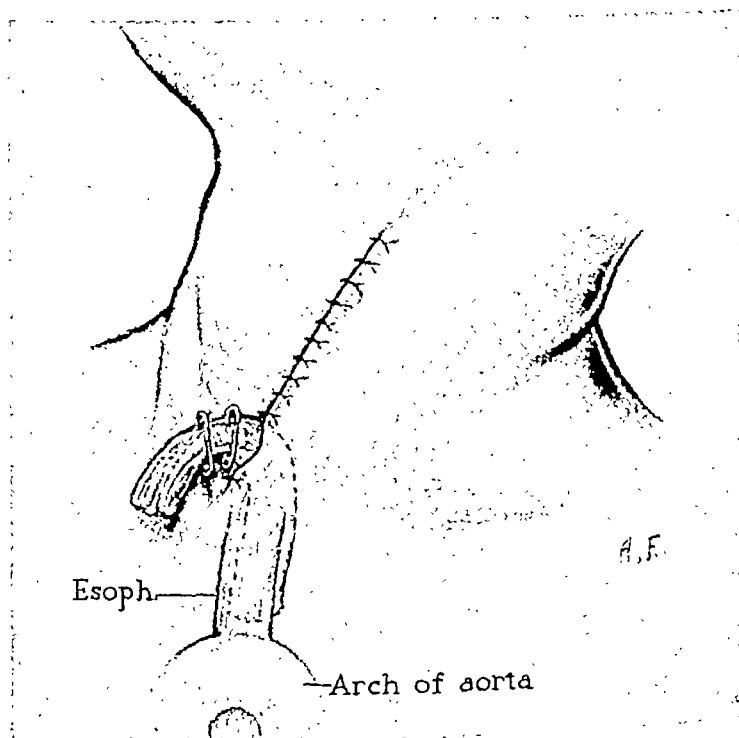


Fig. 3.—Completed operation. Gauze emerging from lower angle of incision.

chest is closed without drainage after inflation of the lung. The packings may be teased out of the stab wound without difficulty after twelve to fourteen days. We believe the latter should be done with the patient anesthetized (intravenous pentothal) (Figs. 4, 5, and 6).

CASE REPORTS

CASE 1 (No. 474037, previously reported¹⁰).—B. S. was admitted to Mt. Sinai Hospital in 1940, with a rather severe hepatitis. He made a satisfactory recovery but later began to have frequent hematemeses. He was readmitted on May 27, 1941, for esophagoscopy after demonstration of esophageal varices in the x-ray films (Fig. 7). After some difficulty

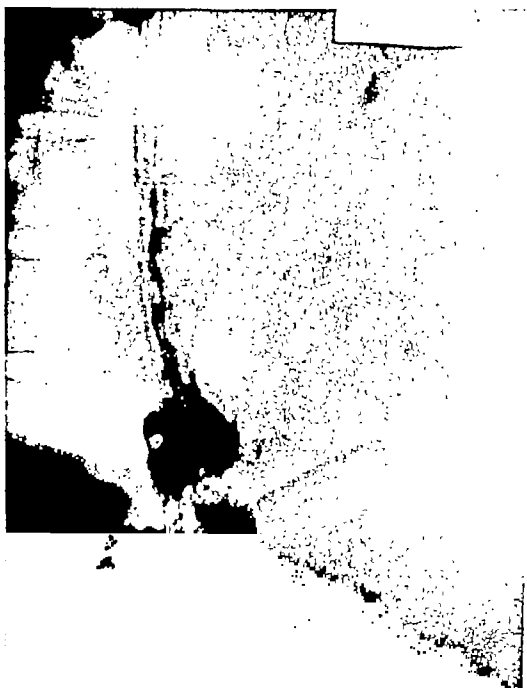


Fig. 7 (Case 1).—Preoperative esophagram showing varices at lower end of esophagus. These were demonstrated on esophagoscopy.

in passing the cricopharyngeus muscle, the tube was introduced and the esophagus examined. Many bulging varices protruding into the lumen of the lower part of the organ were visualized (M.L.S.). The patient, following withdrawal of the instrument, complained of severe interscapular pain. A roentgenogram showed air in the upper posterior mediastinum, confirming the clinical impression of instrumental perforation. A cervical mediastinotomy was performed through a right-sided incision by another surgeon. A large irregular per-

the end of which emerges from the lower angle of the cervical incision. The latter is sutured throughout most of its extent. The packing is removed after twelve to fourteen days without much difficulty (Figs. 1, 2, and 3).

The operation of packing the lower posterior mediastinum is one of greater magnitude and requires meticulous dissection. A long incision is made over the right seventh rib which is resected subperiosteally. The chest is entered and a rib-spreader is inserted, thus affording a clear view of the entire mediastinal pleura overlying the esophagus. A longitudinal incision is made along the

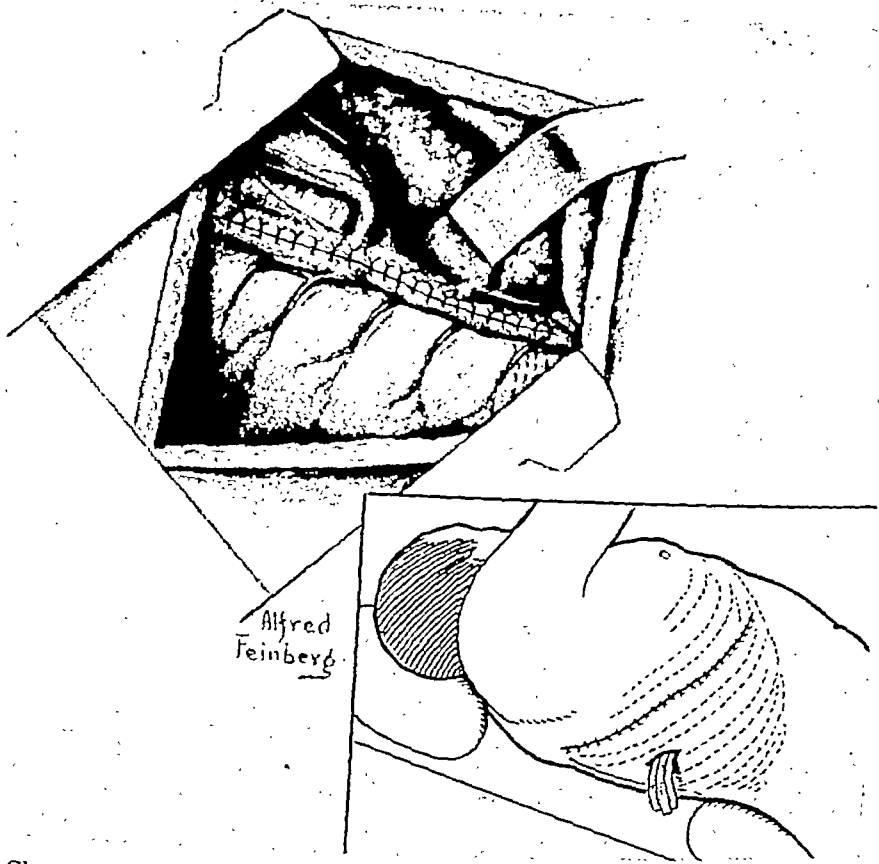


Fig. 6.—Closure of mediastinal pleural flaps over gauze packings. The latter are now completely extrapleural and emerge from the chest as indicated in the insert.

mediastinal pleura, care being taken not to injure the azygos vein. The flaps of pleura should be preserved for later use in closure. The esophagus is now carefully freed from its mediastinal bed from the aortic arch to almost the diaphragm. In accomplishing this maneuver it may be necessary to ligate a few small veins in the mediastinum. Three strips of one-inch single-thickness gauze packing are placed longitudinally along the esophagus. A channel is now bluntly dissected between the parietal pleura and the rib cage and the long ends of the three packings are withdrawn beneath the lateral flap of mediastinal pleura into this channel and brought out extrapleurally through a stab wound in a subjacent intercostal space. The incision in the mediastinal pleura is now repaired, thus effectively covering the esophagus and the gauze packings. The

of mediastinal packing. At this time the hemoglobin was 48 per cent and the red blood cells numbered 3 million. The only positive laboratory test was a one plus cephalin flocculation. Esophagoscopy disclosed numerous varices in the lower third of the esophagus, but most prominent in the region of the cardia. The esophagram was confirmatory.

Packing of the posterosuperior mediastinum was done on Dec. 7, 1945. The packings were removed twelve days later and the patient was discharged. He was well until February, 1946, when he passed a large black stool and hemoglobin dropped to 34 per cent. One transfusion of 500 c.c. of blood brought an immediate response. He was readmitted on April 22, 1946, following another hemorrhage. Hemoglobin this time was 65 per cent. Esophagoscopy at this time disclosed numerous fibrotic varices around the cardia. It was assumed that the bleeding had its origin in varices of the stomach. He gained forty pounds and returned to work. When seen on Jan. 20, 1949, three years after operation, he stated during this time he had had six episodes of tarry stools each lasting about a day. There had been no repetition of the previously severe hematemesis. It was at this time that we had decided to extend the operation to include packing of the lower mediastinum and it was therefore suggested to the patient that this operation be carried out in his case. He accepted with alacrity and was accordingly readmitted on March 3, 1949. The operation was performed on March 8, 1949, by the technique already described. An important observation at this time was the finding of an extraordinarily extensive network of vessels in the periesophageal area above the arch of the aorta, the site of the first packing operation. In the mediastinum below the arch, there were a few veins which required ligation for proper mobilization of the esophagus.

Note.—From the history, there is the strong probability that this patient's illness was traumatic in origin and resulted in a splenic vein thrombosis. The history also indicates a large alcoholic intake during the years 1942 to 1944. However, there has never been any evidence of liver damage to warrant a diagnosis of cirrhosis. It is, of course, too soon to make any statement concerning the effect of the last operative procedure, but there can be no question about the abatement of hematemesis following the original packing procedure.*

CASE 3 (No. 575135).—P. S., aged 26 years, was admitted on Jan. 8, 1948, with the story that nine years before, following a severe hematemesis, he was operated upon at another hospital and his spleen was removed for "early Banti's disease." Nine months later he had another severe hemorrhage and it was about at this time that extensive varices were demonstrated by esophagoscopy. During the succeeding seven years he had one or two episodes each year consisting of nausea, severe hematemesis, and melena requiring multiple transfusions. In the past year the attacks were more frequent and more severe. Examination was negative except for the abdominal scar. Liver function tests were negative and the stool contained no blood. The hemoglobin was 47 per cent. The polymorphonuclear leucocytes numbered 6,050, but the differential count was normal. The platelets were 400,000. Bone marrow studies revealed normoblastic marrow. X-ray examination of the esophagus disclosed extensive varices involving the lower third of the organ.

Packing of the superior mediastinum was done on Jan. 13, 1948. Convalescence was without incident and the packings were removed on the fourteenth day. The patient was discharged on January 28.

Follow-up observations.—On his first return one month later, he stated that two weeks after leaving the hospital he had had an episode of bleeding requiring three transfusions. He recovered quickly. On April 29, 1948, he stated that he had had no further bleeding and was gaining weight and had returned to work. When seen on Oct. 31, 1948, he stated that there had been noted during the preceding two weeks a drop in his hemoglobin to 48 per cent without, however, any evidence of bleeding. The stools were repeatedly guaiac negative. It was thought that some type of blood destruction was taking place to explain

*This patient has remained well to date, February, 1950, with no further episodes of hematemesis or tarry stools.

foration just below the hypopharynx was sutured loosely and several gauze packings were inserted downward into the mediastinum. The patient made a satisfactory recovery and has remained well since. He has had no bleeding in the succeeding eight and one-half years. Recent esophagrams show disappearance of the varices (Fig. 8).

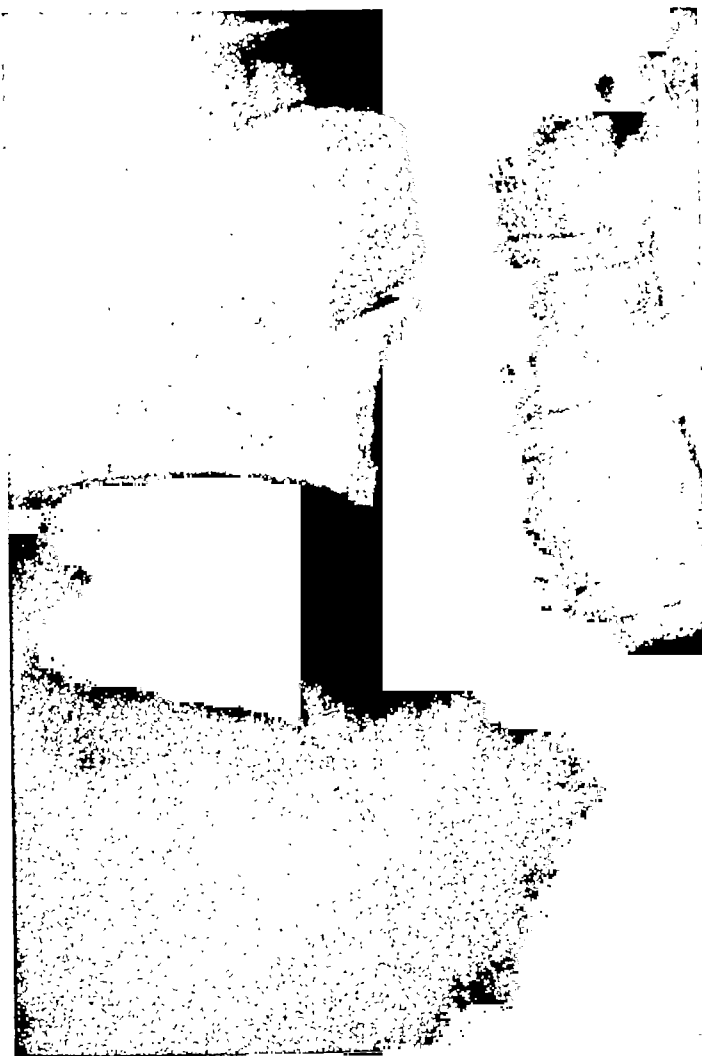


Fig. 8 (Case 1).—Esophagram taken one year postoperatively showing no demonstrable varices. Esophagoscopy was not repeated for obvious reasons. (See Case Report.)

CASE 2 (No. 548020, previously reported¹⁰).—E. R., aged 21 years. At the age of 13 years, this patient suffered an injury to his spleen and subsequently had several episodes of hematemesis and tarry stools. In 1942, after a thorough study at Bellevue Hospital, a diagnosis of Banti's disease was made and splenectomy was performed. Six months later, the hematemesis returned and he was referred to this hospital for injection therapy of the varices. The first admission was on April 14, 1943. Esophagoscopy disclosed extensive varices at 38 cm. from the incisor teeth. Sodium morrhuate solution was injected.

A second injection was given on May 13, 1943. He remained well until August, 1943, when he had another severe hemorrhage and his hemoglobin dropped to 10 per cent. He was readmitted for repeated transfusions and was discharged two weeks later.

During 1944, he had many morrhuate injections, but the bleeding continued with increasingly shorter free intervals. He was finally admitted on Nov. 26, 1945, for the operation

During the following year, the condition remained satisfactory until Nov. 4, 1947, when he had a severe bleeding episode, vomiting a large amount of liquid and clotted blood. He was readmitted on this day suffering from acute blood loss. The hemoglobin was 5.7 Gm. Following repeated transfusions and after it was evident that bleeding had stopped for ten days, the posterolateral mediastinum was packed through a cervical incision on Nov. 8, 1947. During the first week, convalescence was very stormy by reason of an extensive bilateral bronchopneumonia. He finally recovered and the packings were removed on the fifteenth day postoperatively. There was no further bleeding and the child was discharged on Dec. 18, 1947.

Follow-up Observations.—On March 4, 1948, two and one-half months later, there was noted a rather extensive collateral venous engorgement over the left shoulder, arm, and antecubital fossa, indicating clearly that an increased load was being thrown on the superior caval circulation. There had been no further bleeding. When seen again on April 1, 1948, the child appeared well and was developing nicely. No bleeding had been noted by the parents.

On Dec. 14, 1948, he was readmitted, following a severe hematemesis, accompanied by tarry stools. The hemoglobin was 4.5 Gm. During the succeeding ten days, he was given 3,000 c.c. of blood. Finally, after it was evident that the bleeding had stopped, packing of the thoracic mediastinum was done on Dec. 24, 1948, by the technique already described. There was noted marked dilatation and thickening of the esophagus. Approximately ten small veins were seen coursing from esophagus to the mediastinal tissues. An attempt was made to preserve these. Two strips of gauze packing were placed about three quarters of the esophageal circumference. He recovered from this operation and the packings were removed under anesthesia on the fourteenth day. He was discharged from the hospital on Jan. 28, 1949.

He was readmitted on Feb. 23, 1949, with the history that four days before he developed an upper respiratory infection. Because of the suspicion of whooping cough, a booster dose of pertussis vaccine had been given. One day prior to admission, following a severe coughing spell, he vomited a large amount of blood. Following admission, bleeding continued for some days requiring repeated transfusions. At the present writing, he is still in the hospital but doing nicely. All bleeding has ceased.

Note.—It is fair to assume that this child's basic disease is a cavernomatous transformation of the portal vein. It is well known that this variety of portal hypertension is particularly difficult to treat surgically and that the results leave much to be desired. It is encouraging to note the extensive collateral circulation over the left shoulder region, suggesting that this was produced by the first packing procedure. It is too soon to state the effect of the second operation. A further period of observation is necessary.*

CASE 6 (580355).—L. L., an Italian man, 60 years of age, was first admitted to the hospital in December, 1947, with the story of repeated hematemesis and progressive ascites of one year's duration. The work-up disclosed esophageal varices and the laboratory findings warranted the diagnosis of moderately severe alcoholic cirrhosis. He was treated medically and finally discharged somewhat improved. The second admission was on May 7, 1948, following a large hematemesis. The abdomen was filled with ascitic fluid for which repeated paracenteses were done. He had repeated episodes of esophageal bleeding requiring multiple transfusions. Finally, on May 25, packing of the superior mediastinum was carried out. Following a normal convalescence, the packing was removed on the fourteenth day and the patient was discharged.

He was well until the morning of his third admission, Aug. 10, 1948, when he vomited a large amount of blood. Examination showed the presence of extensive ascites and, by laboratory tests, falling liver function. He continued to bleed for eight days, requiring

*As of March 1, 1950, the patient continued to do well. There was one severe bleeding episode during the latter part of 1949.

this episode. He was last seen on Jan. 20, 1949. He stated that two weeks before he had coughed up without effort a small amount of blood. This was the only episode of bleeding in a year. His general condition was excellent and the hemoglobin was 80 per cent.

Note.—This patient's portal hypertension is probably of extrahepatic origin. That there has been marked amelioration of the symptom of hematemesis there can be no doubt. A longer period of observation is necessary before a final appraisal can be made.*

CASE 4 (No. 572631).—W. B., aged 61 years, was admitted on Nov. 11, 1947, as an obvious case of rather severe alcoholic cirrhosis of three years' duration with frequent episodes of pallor, dyspnea, dizziness, abdominal cramps, and the passage of tarry stools. He had been a patient at Bellevue Hospital and was referred for the packing operation. Examination showed a pale, hyperpneic, chronically ill, elderly man. The liver was enlarged, hard and regular in outline. The esophagram disclosed extensive varices involving the lower one-half of the organ. At esophagoscopy there were seen numerous varicosities starting at 28 cm. from the incisor teeth and extending downward to the lower end where a rosette formation was found. The blood picture disclosed a severe secondary anemia with a hemoglobin of 43 per cent.

After preliminary therapy consisting of proper food intake, vitamin administration, and transfusions, packing of the superior mediastinum was done on Nov. 18, 1947. Convalescence was uncomplicated except for a mild pulmonary infection which yielded to penicillin therapy. The packings were removed on the fourteenth day and the patient was discharged soon after.

Follow-up Observations.—Two months later he stated that there had been no further bleeding. He had gained weight and had returned to work. The liver seemed to be larger. He was readmitted on Jan. 31, 1948, following the passage of a large tarry stool. The bleeding stopped following transfusion and he was discharged on February 21. On July 15, 1948, he stated there had been no further bleeding and that he felt well. On Aug. 22, 1948, he was admitted for the third time following the passage of a large tarry stool the day before. The liver was now markedly enlarged. The hemoglobin was 9.5 Gm. and the red blood cells 2,690,000; urea nitrogen, 21; total protein, 5.3; icteric index, 3; alkaline phosphatase, 8 k.a.; thymol turbidity, +; Cephalin flocculation, +. Prothrombin time was eighteen and one-half seconds (control, fifteen). He was treated with repeated transfusions. He was finally transferred to Bellevue Hospital because of the development of psychotic symptoms. According to our latest information he is at present a patient in Presbyterian Hospital.

Note.—This is a case of moderately severe cirrhosis of the liver of alcoholic origin with portal hypertension. As time goes on and experience increases it may be found that, in cases of severe portal hypertension due to intrahepatic block (cirrhosis), the packing procedure will have no effect on the gastrointestinal bleeding coming from the greater splanchnic area. When the present-ing symptom is hematemesis, then the packing operation should, at least, be tried first.

CASE 5 (No. 572322).—R. C., a child 2½ years of age, was first admitted to the hospital in October, 1946, because of fever and oral ecchymoses of one month's duration. Examination disclosed splenomegaly, slight hepatic enlargement, thrombopenia, leucopenia, and anemia. X-ray studies indicated extensive varices involving the entire thoracic portion of the organ. During this stay, frequent tarry stools were noted.

He was readmitted in November, 1946, because of frequent severe hematemesis and the passage of tarry stools. Following repeated transfusions, splenectomy was done. The spleen revealed evidence of congestive splenomegaly. Convalescence was uneventful and he was discharged.

*When last seen in December, 1949, this patient was well and had had no further bleeding episodes.

where study revealed severe anemia, splenomegaly 10 cm. below the costal margin, extensive esophageal varices, and a duodenal ulcer. Again splenectomy was attempted but the operation was abandoned because of extreme vascularity.

After two months she was discharged for out-patient care where a mild diabetes was discovered. One year previously an active duodenal ulcer was disclosed on x-ray examination. She continued to have periodic hematemesis. In the year prior to our examination she was admitted to New York Hospital on four occasions for massive bleeding, requiring on each admission 5,500 c.c. of blood. The last admission was six weeks prior to the writing of this paper and her condition was so precarious that she required 700 c.c. of blood daily for ten days for bare maintenance. At the time she was transferred to Mt. Sinai Hospital her hemoglobin was 13 Gm.

Examination revealed a thin, poorly nourished woman. There was a loud, long systolic murmur over the precordium. The spleen was felt six fingers below the costal margin and the liver three fingers. There was no ascites. Hemoglobin was 13.4 Gm.; platelets, 40,000; prothrombin time fourteen seconds (control thirteen and five-tenths); cephalin flocculation +. A gastrointestinal x-ray series disclosed large tortuous varices in the lower third of the esophagus and a deformed duodenal bulb.

On the eleventh day of admission she began to bleed again and during the succeeding five days required 3,500 c.c. of blood. Finally, on Nov. 23, 1948, packing of the superior mediastinum was carried out. The esophagus was bluntly separated from the prevertebral fascia behind and from the trachea in front as far down as the aortic arch. Two pieces of packing were inserted practically encircling the esophagus. Convalescence was uneventful and the packings were removed on the fourteenth day. Because of some hoarseness, laryngeal examination was done and disclosed a fixation of the left vocal cord, presumably due to left recurrent nerve injury. She volunteered the information that she had not felt so well for three consecutive weeks in the preceding five years. She was discharged on Dec. 20, 1948.

She was readmitted to the Medical Service on Jan. 1, 1949, following a small hematemesi which abated promptly. She was jaundiced, and moderately ill. After thorough study, it was felt that she had homologous serum jaundice of viral origin following one of her many transfusions. She improved rapidly and was discharged on Feb. 20, 1949. When last heard from, March 17, 1949, she had had no further bleeding and, aside from ankle edema, felt well.

Note.—This is a most unusual case of portal hypertension of extrahepatic origin extending over a period of forty-one years. Two attempts at splenectomy were abandoned because of extensive intra-abdominal venous collateral channels. There has been marked amelioration of the symptom of hematemesi following packing of the superior mediastinum.*

COMMENT

To recapitulate, the patient in Case 1 has remained well eight and one-half years. Whether he would have stopped his bleeding episodes without the packing procedure is a moot question. The fact remains that varices were demonstrated before operation and were not evident upon x-ray examination one year later. The second patient has had no further hematemeses for three years. The cause of the tarry stools is probably bleeding from cardiac or gastric varices. Whether or not the more extensive packing procedure will influence this, further observation alone will tell. The third patient has been markedly improved by the operation. The fourth patient with alcoholic cirrhosis has shown little or no improvement. The fifth patient, the child with cavernomatous transfor-

*It has been learned that the patient died at Bellevue Hospital on Feb. 17, 1950. The details of the last illness could not be obtained in time for publication.

repeated and large transfusions. The hemorrhage finally ceased, and his condition improved. X-ray studies revealed extensive esophageal varices extending into the stomach. He was finally discharged on September 16.

He remained relatively symptom free, except for the ascites, until Feb. 23, 1949, when he was admitted following an exsanguinating hemorrhage. The bleeding continued, did not respond to multiple transfusions, and the patient died on March 5, 1949.

Note.—As in Case 4, this was an example of severe portal hypertension due to hepatic disease (cirrhosis) with ascites. The packing procedure had no effect on the bleeding or ascites. Perhaps in such instances, in the future, packing of the thoracic mediastinum should be undertaken either simultaneously with or soon after the cervical procedure. This warrants further trial.

CASE 7 (No. 581351).—E. S., a woman 49 years of age, was admitted on May 23, 1948, with the following history. Eleven years before, following the development of epigastric pain and the finding of an enlarged spleen, she was operated upon in another hospital and a thrombosis of the splenic vein was disclosed. The spleen was not removed. X-ray examination of the gall bladder revealed calculi. The patient had previously had an attack of obstructive jaundice thought to be due to a stone. A diagnosis of polycythemia was made seven years previously and treatment consisted of phlebotomy and x-ray therapy to the splenic area. Three months before admission she experienced her first massive hematemesis. This was repeated several times and required multiple transfusions.

During the period of radiotherapy to the spleen, the patient was in the hypochromic phase of polycythemia. Her gall bladder symptoms responded to dietary restrictions. Examination disclosed a large spleen extending three fingers below the costal arch. Aside from a mild anemia, the blood picture was negative. The esophagram disclosed several large varices at the lower end of the organ.

Packing of the superior mediastinum was carried out on June 16, 1948. Convalescence was uneventful and the packings were removed on the thirteenth day. She was discharged on June 30, 1948.

We have not seen this patient since she left the hospital and moved away. We learned recently (March 15, 1949) that, since discharge, she has had two episodes of esophageal bleeding requiring transfusions and that, in February, splenectomy had been performed in Chicago.

Note.—This is a case of portal hypertension caused by extrahepatic block, thrombosis of the splenic vein. The packing of the superior mediastinum apparently had little effect on the symptom of hematemesis.

CASE 8 (No. 587914).—This is perhaps the most fascinating and dramatic case in the series. A. P., a woman 62 years of age, was transferred from New York Hospital* on Nov. 5, 1948. She had suffered from repeated hematemeses over a period of forty-one years, the onset being four months following the birth of her first child. At that time a large spleen was disclosed. Thirty-two years before this transfer splenectomy had been attempted† but the operation could not be consummated because of extraordinarily extensive venous channels. X-ray therapy was carried out without improvement. The hematemeses occurred two to three times a year and required transfusions. Fifteen years prior to admission, following the menopause, the frequency of the hematemeses increased to two a month, each averaging two to three cupfuls of blood. About this time she noted the onset of epigastric pain with the bleeding episodes. Two and one-half years before transfer, following a particularly severe bleeding bout, she was admitted to the New York Hospital

*Through the efforts of Dr. Edwin Hauser.

†By Dr. Allen Whipple.

PULMONARY ALVEOLAR ADENOMATOSIS

CANCEROUS PULMONARY ADENOMATOSIS, ALVEOLAR CELL CARCINOMA OF THE LUNG, JAGZIEKTE?

H. BRODIE STEPHENS, M.D., AND SIDNEY J. SHIPMAN, M.D.
SAN FRANCISCO, CALIF.

WOOD and Pierson⁷ reported a case of pulmonary alveolar adenomatosis in March, 1945, the diagnosis having been made before death. The diagnosis was established in their patient by lobectomy. This operation was performed* six months prior to the patient's death. It was because of the interest aroused by this patient's illness and the fact that one of us (S. J. S.) had followed the case with Dr. Pierson, that the diagnosis of pulmonary adenomatosis was seriously considered prior to operation in the case to be reported.

Since the presentation of our paper at the Meeting of The American Association for Thoracic Surgery in March, 1949, at New Orleans, two important contributions on the subject of pulmonary adenomatosis have appeared in the literature (Delarue and Graham¹ and Swan⁶). Delarue and Graham reported the presence of an alveolar cell carcinoma in a man aged 35 years who had symptoms for approximately twelve months prior to admission to Barnes Hospital. A right lower lobectomy was performed in June, 1943, since the disease appeared grossly to be confined to this lobe. There was no evidence either at operation or pathologically of involvement of the regional lymph nodes. Microscopically, the predominant feature of the lesion was the tendency to reproduce the alveolar structure. Some areas of lung tissue showed complete replacement of the alveolar linings by hyperplastic epithelium. At the periphery of the lesion, there were occasional isolated groups of alveoli where the epithelium was hyperplastic and tended to grow in small papillomatous formations projecting into the lumen of the alveoli. In still other areas, the tendency to the reproduction of alveolar structure was not seen, the cells going wildly in clumps and cords in a loose fibrous tissue stroma. In these zones, the malignant cells showed marked variation in size, shape, and staining reaction, and mitotic figures were fairly frequent.

Delarue's and Graham's patient remained well for over three years, and in October, 1946, an x-ray examination of the chest showed a lesion overlying the right diaphragm. Right pneumonectomy was performed in December, 1947, four and one-half years following lobectomy. Actual invasion of the chest wall was present at this time. The mediastinal lymph nodes were apparently negative. The microscopic pattern of the tumor found at the second operation was very similar to that seen at the first operation. When last seen (September, 1948), the patient was in excellent clinical condition.

From the Department of Surgery and Department of Medicine University of California Medical School.

Read at the Twenty-ninth Annual Meeting of The American Association for Thoracic Surgery, New Orleans, La., March 29-31, 1949.

*By Dr. Emile Holman.

We used iodoform gauze in the first few cases. The patients developed severe temperature reactions and we attributed it to the absorption of iodoform and gave it up for that reason. I think more irritation, of course, can be produced by iodoform than with plain gauze.

Dr. Sweet, I do not know how long we will have to wait; I do not know whether Dr. Blakemore knows how long he will have to wait with his portocaval shunts. This is presented as a substitute minor operation, and I think should be tried first, before portocaval anastomosis. However, I think the period of time between cervical mediastinotomy and posterior mediastinotomy should be lessened. I believe if a patient does not respond within three or four months he should have the posterior mediastinum packed as a second stage. We have had no mortality in the two cases we did, but that is a small group.

We went into the question of Gelfoam and cellulose and all the other irritating substances, but I think the experimental work done in other clinics indicates that when irritating substances which do not have to be removed are introduced, a large fibrous tissue element is formed, with a minimum of venous channels and a large scar tissue replacement. For this reason we have not used the substances mentioned.

However, experimental infection has been generally unsuccessful. In addition, Dungal stated, in 1946, that he had not seen jagziekte in man, although many of the shepherds were in daily contact with sick sheep, often in closed houses for long periods of time, and hence every opportunity for contracting the disease was present.

The ovine disease is very insidious in its onset. It runs a chronic course so that weeks may elapse before its presence may be suspected even by an experienced observer. If the disease has not reached an advanced stage, a flock at rest containing many affected sheep may be looked at most carefully without any sickness being noticed. There is no loss of appetite, and but little loss in condition until the latest stage when the sheep are apt to become very emaciated. A cough, at first dry, is ultimately associated with the development of moist râles on auscultation. Eventually, the condition becomes productive of much secretion, at first thin and frothy which pours out of the nostrils when the sheep inclines its head towards the ground. Until or unless a terminal secondary infection sets in, the disease characteristically runs an afebrile course.

CASE REPORT

The patient was a white, married woman. She was 51 years of age at the time of admission to the University of California Hospital on April 18, 1948. Family history and marital history were noncontributory. The past history was of interest in view of the fact that in 1939 or 1940, her neighbors had a small flock of sheep. The flock became ill with an acute type of pulmonary infection from which many of the sheep died. The patient's contact with the sheep was casual, involving an interest in the misfortune of her neighbors, but not to the extent of any care of the sick animals. The patient, at no time, actually handled the sheep, but she was frequently only a few feet from them, and at times when the weather was windy and dusty. It was about this time, or shortly thereafter, that the patient noticed the appearance of a dry, hacking cough that was irritative and nonproductive. Some time between 1941 and 1942, the patient began to raise small amounts of clear, mucoid, frothy sputum accompanied by an increase in the severity of the cough. A physician was consulted because of these pulmonary symptoms. Physical examination was essentially negative, but an x-ray examination of the chest was said to show some abnormal findings in the region of the right lower lobe. Unfortunately, this roentgen film was lost and cannot be compared with subsequent roentgenograms. The provisional diagnosis at this time was bronchiectasis involving the right lower lobe. There was some progressive increase in the severity of the patient's cough from 1942 to 1945. The sputum remained mucoid and frothy, although increased in amount. A roentgen film of the chest dated June 6, 1945, showed a triangular area of increased density in the right lower lung field immediately above the diaphragm and posterior to the area of the interlobar fissure between the right middle and lower lobes (Figs. 1 and 2). Bronchoscopy at this time (June, 1945) was negative. In addition, the sputum now became thick, yellow and foul-smelling, and the patient complained of her sputum having a disturbing musty odor. There was some vague right lower chest pain accompanying this change in the quality of the sputum, but the pain was at no time typically pleuritic. Roentgen films of the chest dated July and December, 1947, revealed an increase in the size of the density in the right lower lung field, the density now being more posterior than in 1945 (Figs. 3 and 4).

Physical examination of the patient at the time of admission revealed a very well-developed and well-nourished woman appearing in good health aside from the persistent coughing. The abnormal physical findings were located in the thorax and pelvis. A slight respiratory lag was noted on the right inferiorly. Fremitus was increased posteriorly at the right. There was slight dullness over the area of the right lower lobe and in this area there were

Delarue and Graham summarized their presentation by pointing out the following: (1) the neoplasm they described represents a specific type of pulmonary tumor that is characterized by a reproduction of the alveolar structure of the lung; (2) the lesion appears to be multicentric in origin; (3) alveolar cell carcinoma is a proper name for the neoplasm they describe; (4) such a neoplasm may demonstrate slow growth, but reappear several years later in a different part of the lungs, and (5) attention is called to the similarity of alveolar cell carcinoma to jagziekte, the South African disease of sheep.

Swan reported under the title "Pulmonary Adenomatosis of Man." He reviewed the cases not included in those collected by Neubuerger and Geever⁴ in 1942, and presented nine cases from the files of the Army Institute of Pathology. Swan called attention to the sex ratio of approximately 1:1 in Neubuerger's and Geever's series, and the ratio of twelve men to fifteen women in the additional twenty-seven acceptable cases that he has collected since 1942. The relative predominance of this condition in the female sex resembles that of bronchial adenoma more closely than that of bronchiogenic carcinoma.

Swan suggested the term "pulmonary adenomatosis" as the preferable designation for so-called alveolar cell tumor of the human lung. If metastases occur, the term should be modified by adding the adjective "cancerous" (cancerous pulmonary adenomatosis). In addition, Swan proposed the following criteria for the diagnosis of pulmonary adenomatosis: (1) alveolar cellular proliferation characterized by the appearance of tall, columnar, mucus-producing cells, (2) absence of an intrinsic tumor of the bronchial tree, and (3) absence of primary adenocarcinoma of any other part of the body.

Swan concluded his summary by pointing out the fact that these tumors appear histologically noncancerous, but must be considered clinically to be cancers since they may kill by local growth or by metastasis. (Neubuerger and Geever estimated that 25 per cent metastasize to the regional lymph nodes and another 23 per cent to distant sites.)

Jagziekte, a South African word from the Dutch, is the name given to pulmonary adenomatosis occurring in sheep. Characteristically, the disease appears when sheep are being driven for some distance and hence the name jagziekte, or driving disease. Pulmonary adenomatosis has been recognized in the sheep of South Africa since 1893, and in addition, has been reported as occurring epizootically in sheep of Iceland, England, and the United States, particularly in certain areas of Montana. Histologically, the usually fatal ovine disease consists of a certain amount of interstitial fibrosis and epithelial proliferations forming papillary intra-alveolar masses which are noninvasive. In common with human pulmonary adenomatosis, there is multicentricity, columnar nonciliated cells, the absence of stromal invasion, and infrequent mitotic figures. Dungel⁵ stated he has never seen in jagziekte metastatic growth in the tracheobronchial glands or elsewhere. No specific agent has been proved to be the cause of the disease, but many investigators have suspected that a virus is the exciting and responsible factor. Transmission of the disease is effected easily by housing healthy sheep with diseased sheep.

coarse râles and wheezing even after coughing. Rectal examination revealed a small fibroid tumor in the fundus of the uterus. (This tumor had been observed by the patient's local doctor for several years and has remained the same size.) The temperature, pulse, respiration, and blood pressure were within normal limits. The only abnormal laboratory finding was a white blood count of 14,150 with 86 per cent polymorphonuclears (85 per cent filamented). Numerous sputum examinations were negative for acid-fast bacilli. Bronchial smears and a single sputum examination were negative for malignant cells. Bronchoscopy and Lipiodol visualization of the right lung were carried out two weeks before lobectomy. The bronchoscopic examination was negative except for possibly some minor stenosis of the right lower lobe bronchus. All the major segmental divisions of the right lung filled normally except for possible mild cylindrical bronchiectasis in the posterior basic division of the right lower lobe.

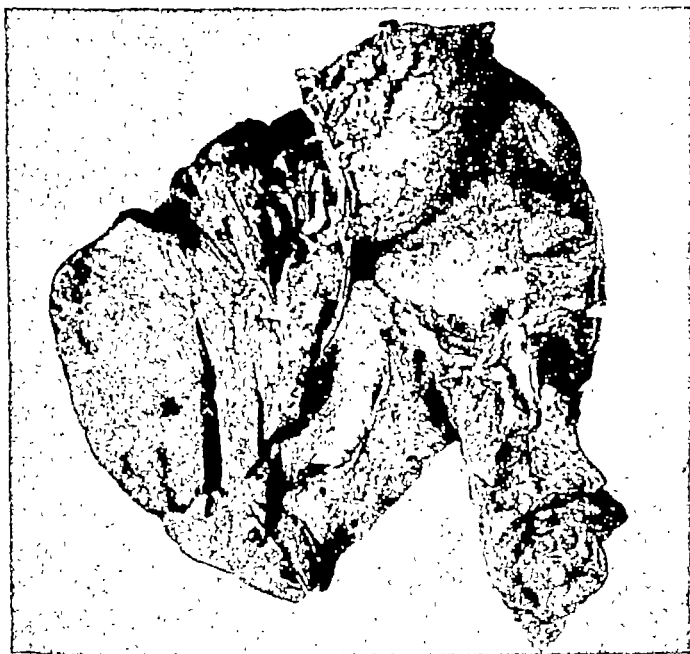


Fig. 5. A.—Gross appearance of the right lower lobe. The consolidated, waxy, gelatinous texture of the lung is depicted in the photograph.

The previous rather casual exposure to sick sheep, the prolonged insidious type of illness, to which no other etiology could be ascribed, plus the resemblance of this patient's history and findings to the Wood and Pierson case resulted in one of us (S. J. S.) entertaining the possible diagnosis of pulmonary alveolar adenomatosis. Exploratory thoracotomy was considered in order, and this was carried out on May 4, 1948 (by H. B. S.).

Operation.—When the incision was made on the right chest, there was no evidence of either pleural effusion or adhesions. The right upper and middle lobes appeared normal in all respects and aerated normally. The appearance of the right lower lobe was unique in the

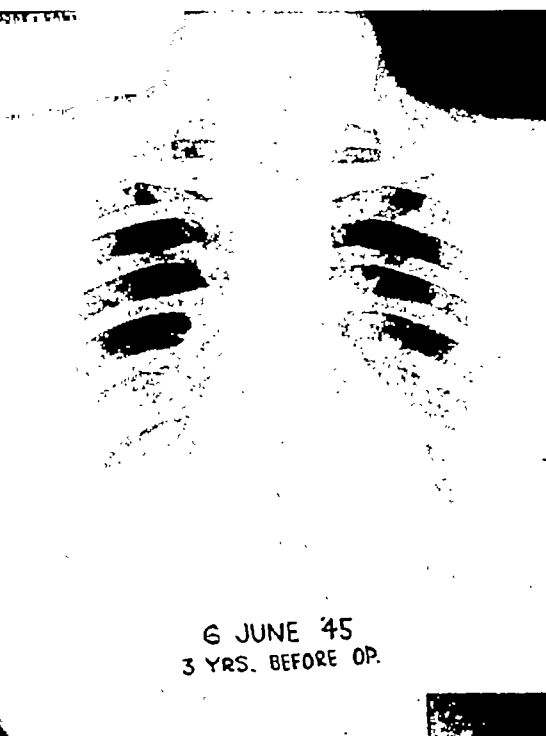


Fig. 1.

Fig. 1.—Anteroposterior projection. The triangular density is present in the periphery of the right lower lung field.



Fig. 2.

Fig. 2.—Lateral projection. The abnormal density is outlined by the two arrows.

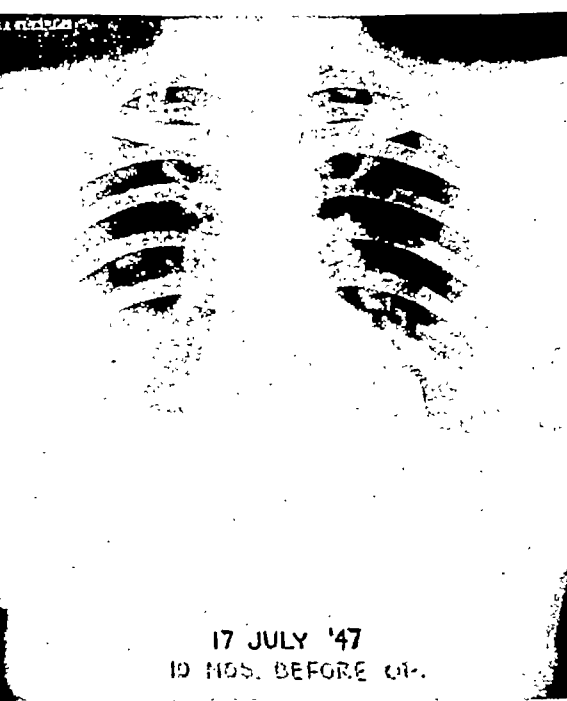


Fig. 3.

Fig. 3.—Anteroposterior projection. The density in the right lower lung field has increased in size.



Fig. 4.

Fig. 4.—Lateral projection. The density in the right lower lung field is more posterior than in 1945.

surgeon's experience. This lobe was quite free of adhesions, but firm throughout. It was salmon pink in color with a peculiar, smooth, solidified surface. The findings suggested that liquid gelatin had been allowed to fill the bronchial divisions of the lower lobe and then hardened (Fig. 5). The operation of lobectomy was readily performed because of the absence of any inflammatory reaction about the lobar hilus.

Fig. 6.

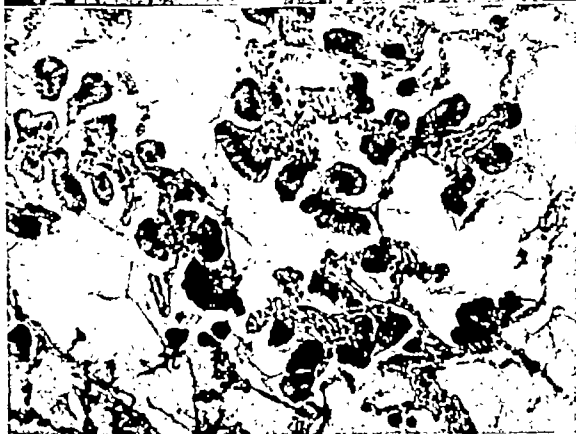
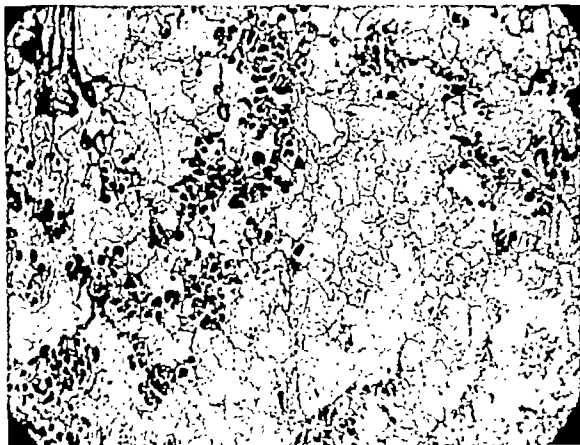


Fig. 7.

Fig. 6.—Photomicrograph of the operative specimen ($\times 22.5$). The apparent multicentricity of the adenomatous epithelial proliferation is well demonstrated. Note the apparently normal alveoli adjacent to the adenomatous proliferations. In addition, note the preservation of the alveolar framework of the lung.

Fig. 7.—Photomicrograph of the operative specimen ($\times 100$). The regularity of the cells forming the adenomatous papillary projections can be seen. There is no suggestion of invasion of the alveolar stroma, nor do the cellular proliferations completely encircle the walls of the alveoli.



Fig. 5, B.—Sagittal section of the gross specimen. The whitish areas (A) represent adenomatous infiltration. The darker areas of the lung in the periphery (B) represent alveoli distended with mucus.



Fig. 5, C.—Sagittal section of the gross specimen demonstrating the confluent nature of a portion of the adenomatosis (A) with a nodular dissemination in the periphery (B). The area marked C represents normal alveoli distended with mucus.

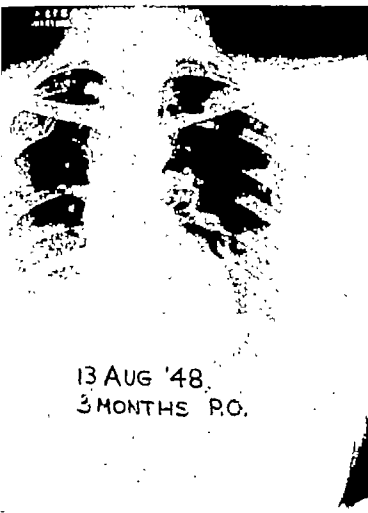


Fig. 9.

Fig. 9.—Anteroposterior projection. There is no evidence of any abnormal density at this time.



Fig. 10.

Fig. 10.—Lateral projection. Compare with Fig. 12. The lung fields appear normal three months following operation.



Fig. 11.

Fig. 11.—Anteroposterior projection. Abnormal density has reappeared in the medial portion of the right lower lung. In addition, there may be increase in the hilar density on the left side.



Fig. 12.

Fig. 12.—Lateral projection. Compare with Fig. 10. The abnormal density is in the lower lung field and posterior.

Pathologic Report.—The specimen consisted of a right lower lobe, weight 470 Gm., with a smooth pleural surface. The lung parenchyma was soft and mushy with a salmon pink color. In some areas the color was pearly white and the parenchyma had a granular appearance, not unlike small granules of cooked tapioca. Often the alveoli appeared to be distended with this mucoid material. A small area of the most superior portion of the lobe showed rare, normal congested lung tissue. The bronchi were free from tumor, and a few small pigmented hilar lymph nodes were present.

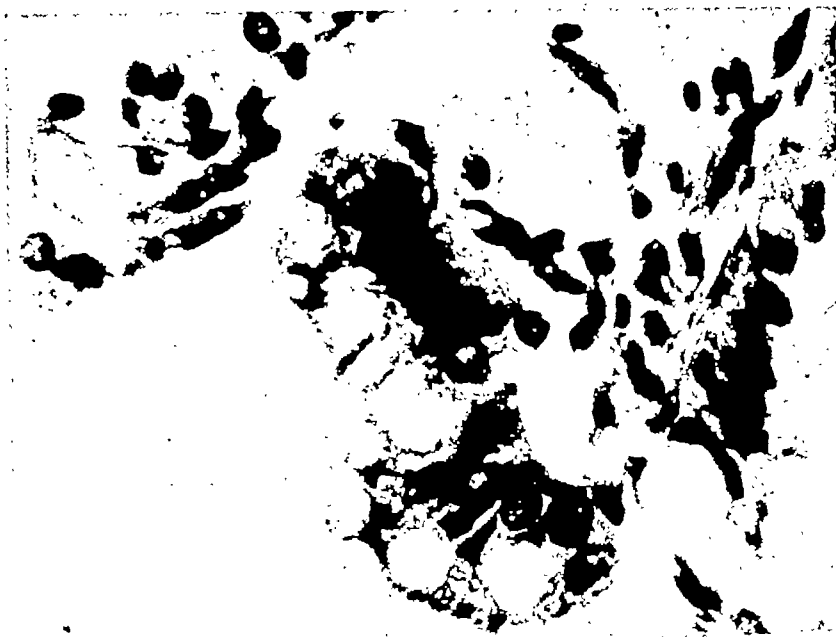


Fig. 8.—Photomicrograph of the operative specimen ($\times 630$). Goblet formation within the cytoplasm of the adenomatous proliferating cells can be seen. Note the regularity of the nonciliated columnar cells with the nuclei located in the base of the cells adjacent to the alveolar border. Appropriate stains demonstrated mucus within the cytoplasm of these goblet cells.

Microscopic Report (Figs. 6, 7, and 8).—The alveoli showed varying degrees of metaplasia and replacement by a simple columnar type of mucus-secreting cell. The cells were somewhat hypertrophic, but well differentiated, and mitoses were uncommon. Varying degrees of differentiation were present, and occasionally well-formed goblet cells were found. The cells frequently had a polypoid appearance. The alveoli were often distended by these cells. The uninvolved alveoli often were distended with mucus and mucoid containing macrophages. Occasional groups of chronic inflammatory cells were noted with early fibrosis of the alveolar walls. The bronchioles were somewhat dilated, and often contained mucus. At the margin of the more normal lung noted grossly, the pulmonary parenchyma was compressed, and the nearby alveoli were distended with large numbers of large reticulo-endothelial cells with finely granular cytoplasm. There were large numbers of eosinophils and considerable intra-alveolar bleeding. The bronchioles and bronchi in this region also were compressed, and the smaller blood vessels showed perivascular cuffing with chronic inflammatory cells and eosinophils. The pleura showed an increased number of chronic inflammatory cells. The hilar lymph nodes showed only moderate anthracosis with a slight increase in the number of eosinophils.

Diagnosis.—Pulmonary adenomatosis.

Subsequent Course.—After fifteen months had elapsed since the operation, the patient remained in relatively good health. She had maintained her normal weight, and had taken several pleasure trips both in and out of the United States. The patient continued to have a mild productive cough, bringing up frothy sputum that was mucoid in character and without odor.* Roentgen films of the chest (Figs. 9 and 10) taken three months following lobec-

*Patient's status continues the same (March, 1950).

SUMMARY

A case of pulmonary adenomatosis has been presented. The patient's pulmonary symptoms began shortly after a definite, though casual, exposure to a flock of sick sheep. The patient was treated by lobectomy. Roentgen evidence of probable persistence or recurrence of the disease appeared seven months following operation. Aureomyein and dihydrostreptomycin in supposed adequate dosage has failed to alter the roentgen appearance of the lungs. The microscopic appearance of the operative specimen resembles more so-called pulmonary adenomatosis than alveolar cell carcinoma.

REFERENCES

1. Delarue, N. C., and Graham, E. A.: Alveolar Cell Carcinoma of the Lung, *J. THORACIC SURG.* 18: 237, 1949.
2. Drymalski, G. W., Thompson, J. R., and Sweeney, H. D.: Pulmonary Adenomatosis, *Am. J. Path.* 24: 1083, 1948.
3. Dungal, N.: Experiments With Jaagsiekte, *Am. J. Path.* 22: 737, 1946.
4. Neuburger, K. T., and Geever, E. F.: Alveolar Cell Tumor of the Human Lung, *Arch. Path.* 33: 551, 1942.
5. Osserman, K. E., and Neuhof, H.: Mucocellular Papillary Adenocarcinoma of the Lung and Lobectomy, *J. THORACIC SURG.* 15: 272, 1946.
6. Swan, L. L.: Pulmonary Adenomatosis of Man, *Arch. Path.* 47: 517, 1949.
7. Wood, D. A., and Pierson, P. H.: Pulmonary Adenomatosis in Man, *Am. Rev. Tuberc.* 51: 205, 1945.

DISCUSSIONS

DR. EVARTS A. GRAHAM, St. Louis.—I am very glad that this material has been presented. I think it of greatest importance, not with regard at the present time to the number of cases, perhaps, but it has very important theoretical possibilities. When I read the abstract, and read that Dr. Stephens apparently thought he had cured his case by lobectomy, I was sorry to see that he had made that statement. I am not surprised to note that in later x-ray examinations he found a recurrence of the lesion because that was to be expected. This is a multicentric disease, there is no doubt whatsoever of that, and because the disease happens to be attacked in a local spot, and that local spot is eradicated, there is no assurance whatever that the patient has been cured of the disease. The evidence indicates that almost certainly it will crop up in remaining portions of the lung.

The most interesting and the most important feature of this disease, however, is that although it seems to be very rare there is plenty of good evidence to indicate that we have known this disease for a good while; but it has been described under various names. At least, that is my concept. Dr. Neuhof, for example, one and one-half years ago in the *JOURNAL OF THORACIC SURGERY*, described this disease under the name of "Carcinoma Mucocellular."

Perhaps Dr. Stephens regards the condition as a benign one since he uses the term "adenomatosis." Actually, the dividing line between what one pathologist might call benign, in this lesion, and what another pathologist will call malignant is so fine and tenuous that really the distinction can hardly be made. If you think that is merely my opinion, I will tell you that at the last meeting of the American Cancer Society in Memphis in January, 1949, this disease came up for consideration. It was the unanimous opinion of the pathologists present at that meeting that this disease was to be regarded as a primary carcinoma of the lung of multicentric origin, arising, I may say, not in a bronchus. It is, therefore, not a bronchiogenic carcinoma as are most cancers of the lung with which we are acquainted, but it arises in the alveoli, in the epithelium of the alveoli. We have always had some doubt in recent years, especially since Fried's work, as to whether there were epithelial cells in the alveoli, but this disease seems to indicate beyond question that there are, and that these cells can develop into a cancer.

tomy revealed normal lung fields. Seven months following operation, an abnormal density appeared in the base of the right lung, and in addition, possible increase in the hilar density of the left lung. The patient was given a therapeutic trial of aureomycin and dihydrostreptomycin. Aureomycin was administered per os 0/5 Gm. every six hours for twenty days, the treatment beginning March 30, 1949. There was no change in the roentgen appearance of the lungs following this course of therapy. Dihydrostreptomycin, 1/0 Gm. daily (intramuscular administration) was begun May 18, 1949, and continued to June 15, 1949, without producing any change in the roentgen appearance of the lungs (Figs. 11 and 12). Bronchoscopy in August, 1949, showed a normal bronchial tree, and bronchial smears at this time were negative for tumor cells.

DISCUSSION OF CASE

It appears from the history, that this patient has had pulmonary disease for at least seven years. Lobectomy has provided a means of determining the pathologic diagnosis, but probably has not altered the course of the disease in that there appears to be progression in the roentgen films without any significant decline in the patient's well-being. This case is similar to the case reports of Osserman and Neuhof² and Delarue and Graham in that there is a history of long duration, an apparent limitation of the disease to a single lower lobe at the time of thoracotomy, and the histopathologic presence of multicentricity, intra-alveolar papillomatous formation, and the absence of regional lymph node involvement.

This case, per se, from the microscopic pathology presented, fits more the criteria proposed by Swan³ for the diagnosis of pulmonary adenomatosis rather than that of Delarue and Graham for the diagnosis of alveolar cell carcinoma. In the case herein presented, under the microscope can be seen: (1) alveolar cellular proliferation characterized by the appearance of tall columnar mucous-producing cells; (2) absence of an intrinsic tumor of the bronchial tree; and (3) absence of primary adenocarcinoma of any other part of the body; thus fulfilling the criteria of Swan for the diagnosis of pulmonary adenomatosis. Delarue and Graham described alveolar cell carcinoma as being characterized by a reproduction of the alveolar structure of the lung; the microscopic appearance of the tumor in our patient fails to demonstrate true reproduction of the alveolar structure of the lung, nor is there evidence of the tumor growing wildly in clumps and cords as was the case in certain areas of the tumor described by Delarue and Graham. The similarity of pulmonary adenomatosis to jagziente becomes particularly interesting in this case because of the patient's casual exposure to a sick flock of sheep shortly before the onset of the patient's pulmonary symptoms. The explanation may well be that the only true relationship between pulmonary adenomatosis and jagziente is simply a specific response of the pulmonary tissue of the two species (man and sheep) to certain nonspecific irritants. As mentioned by Drymalski and associates⁴ this hyperplastic response of the pulmonary epithelium may eventually cause death by extensive involvement of lung parenchyma (jagziente) or by progressing into a true malignancy (alveolar cell carcinoma).

time, one cup. The diagnosis at that time was pneumonitis with rupture of a sizeable vessel. From this pneumonia he is said to have made a recovery, but not completely because the dyspnea was still present, and in addition he had progressive pain throughout the chest. He was not incapacitated, however, and was able to carry on his occupation in a meat and grocery store.

In February, 1948, he did get to an extremity and was again studied in Chicago, where a diagnosis was made of carcinoma of the lung and x-ray treatment was advised in view of the long history. It was said that he improved somewhat as a result of x-ray treatment; he felt so, subjectively, and when I saw him his first statement was, "Under no circumstances will I allow you to do a radical operation upon me. I am interested in x-ray treatment." But it did not seem to me, that this diagnosis of cancer was completely established in spite of the fact that tubercle bacilli were not found. The sputum did not show evidence of carcinoma in the cells, and by roentgenology no differential could definitely be made of this lesion from either fibroid tuberculosis or a suppurative process, bronchiectasis or abscess without fluid level, or perhaps even fungus disease.

At any rate, he allowed me to carry out an exploratory operation for the purpose of diagnosis, and when I opened the chest I found much the same appearance that Dr. Stephens has presented. The upper lobe was entirely solid except for a periphery of aerated lung, I would estimate 1.5 cm. in width; the lower lobe had several nodules in it. When the lung was sectioned it looked like a fungus infection or, as Dr. Stephens has suggested a gelatine transplant in lung tissue. The pathologist at our hospital, Dr. E. L. Heller, who is Associate Professor of Pathology at the University of Pittsburgh, made slides for me which I have the opportunity to show.

I would like to show first the roentgenologic series; I was able to get the picture taken in 1946, and follow the case roentgenologically until the present (Slides). This was in April, 1946, at the time he developed the original pain; the picture on the left was taken in April, 1947, one year later. It was stated to him that it showed no definite change from the picture of April, 1946, that the shadow noted was not significant of any trouble. I believe the disease could be demonstrated definitely at that time. The next pictures were taken in September, 1947, and October, 1947, at the time he said he had the episode of pneumonia. This was taken when he had made a symptomatic recovery. It was stated to him that there were some changes in the pleura and a certain amount of residue in the lung. The next was taken in February, 1948, in Chicago, when he was studied. No bronchoscopy was permissible because of the absolutely rigid neck. When I came to have bronchoscopy done Dr. McCullough said he had never failed to get a bronchoscope down at least into the trachea and upper bronchial tree, but he failed in this instance.

This picture was taken in January, 1949. I would feel that it is impossible from that roentgenologic appearance to make a certain diagnosis. Next is the low power appearance of the biopsy from the lung; a very widespread lesion, the alveoli are filled or almost so with these papillary projections; in this particular field there is only part of the entire lung that is not completely involved. This is the medium power presentation and shows a tremendous amount of mucin and a number of desquamated cells thrown into the alveoli. The high power picture is next. On this basis Dr. Heller felt we were dealing with malignancy definitely ingrafted on an originally benign adenomatosis. He made the diagnosis not on the basis of the irregularity of the cells so much as on the hyperchromatism in the alveoli and the fact that the cells were getting quite large and perhaps displacing one another.

I would say that the prognosis in this disease is quite definitely bad. So far as I can judge from any reading, the life expectancy from the time of development, and we do not know when it develops really, is not more than two or three years. Obviously, from the discussion already presented, surgery is not the answer. I am intrigued with the suggestion that perhaps if the disease can be diagnosed at an early time, antibiotics might have some influence against the possible virus that may be basically in the picture. I still think it is almost a mystery disease.

One of the first descriptions of this disease, worked out in sheep, was made by Cowdry, of St. Louis, about twenty-two years ago. He was asked by the South African government to investigate this epidemic. At that time the disease was not known anywhere else than in South Africa. Now it has spread around the world and has involved the sheep in the United States. It is known to the sheep men by the very noneuphonious name of "snotty-nose" which characterizes the discharge. It is interesting that in Dr. Stephens' case, the human case, this same symptom was present. There seems to be little doubt about the contagiousness of the disease as it affects sheep, although inoculation experiments so far have been unsuccessful. Could it be possible, therefore, that we have now confronting us, just becoming recognized, a form of cancer of the lung which really is a contagious disease? Something which is quite different from anything that we have known about heretofore, but which probably is not nearly so infrequent as we have ordinarily thought.

In 1942, I operated on a man who had, in the right lower lobe, this disease. I did a lobectomy, and had the same difficulty at the operating table in deciding what the lesion was. Because of war conditions and depletion of staff it was impossible to get a frozen section at the operating table. I decided it was merely an inflammatory disease. I took out the lobe, and he was perfectly well for three years after this procedure. In 1946, he came under the care of Dr. Ashmore of Wichita, Kan., who found that the remaining portion of the lung was extensively involved and he advised removal of the rest of the lung, but it was not until 1947 that the patient permitted Dr. Ashmore, who is here this morning, to take out the rest of the lung. The microscopic examination of the removed portion was identical with what I had removed five years before. The other lung seems to be in satisfactory condition at this time, but there is hardly any doubt, in my mind at least, that before long the other lung will also show the same lesion.

There is one other point that is most interesting about the whole picture; that is, that this disease which you have seen portrayed this morning in the slides, is not very different from, in fact in many respects it is identical to, mouse cancer of the lung which is really a multiple malignant adenomatosis. The prevailing opinion has been that this condition is due only to genetic factors. If I had more time I would bring to your attention some reasons for thinking that actually in this inherited disease of mice which has been thought to be due merely to genetic factors we are probably dealing with a condition very similar to—I will not say identical with—but very similar to jagziekte. A virus infection, perhaps the same virus, may be responsible for both conditions.

DR. H. R. DECKER, Pittsburgh.—Dr. Stephens has brought us this presentation in a very important way, supplemented by Dr. Graham's remarks, which leaves me little to say except to present a case report to which I was introduced only three weeks ago. I think, in view of the opinion at the present time, that we might almost call this a mystery disease; certainly there is nothing in pulmonary pathology quite like it. At first these cells are orderly, there is no invasion of stroma, there are practically, in cases so far reported, no metastatic lesions. I think in the literature to which I have had access, two cases had mediastinal lymph node metastases, but in a very late situation and found at post-mortem. The importance of Dr. Stephens' presentation lies in the fact that the disease was recognized before post-mortem, and my case is in the same category.

Briefly, this man, 58 years old, was shot in the chest in 1919, in a hunting accident and had a few shrapnel fragments left in the lung. In 1934, he had a very serious dental infection with osteomyelitis which gave him almost a rigid cervical spine. The significance of that will appear in a moment. In 1940, he had double pneumonia treated with sulfonamides, from which he said he made a very satisfactory recovery and was asymptomatic within six months. In 1946 following exposure to a flock of sheep ill with jagziekte, he developed pains through the chest of an irregular type, not constant, and was examined roentgenologically. It was suggested that the pain might be due to late effects of the few shrapnel fragments left therein. He developed progressive dyspnea and, in September, 1947, had a hemorrhage, rather severe, for which he was hospitalized, and within three days he had a second hemorrhage; it was said that the amount of blood lost the first time was one and one-half cups, and the second

of the Archives of Pathology, which will soon appear. Of the nine, eight were diagnosed at the time of autopsy. The other patient, who had a lobectomy, is still alive but shows definite evidence of recurrence in both lungs.

The two additional cases recorded at the Institute of Pathology are not completely reported. One was diagnosed at autopsy, and one was the patient I have reported, who remains well seven months after surgery.

DR. ROBERT T. FOX, Hines, Ill.—I wish briefly to report a previously published case, presented by the pathology department of the Municipal Tuberculosis Sanitarium in Chicago. Three patients were reported upon, one of which had right lower lobectomy. That patient died one and one-half years later of multiple distant metastases showing the same histologic picture as the primary lesion.

We at the Veterans Administration Hospital at Hines, Ill., recently had a case which differs from the others in that it was a fairly rapid course, some 6 months from onset of symptoms to death. The predominant symptom was the marked bronchorrhea, amounting to some three or four quarts of watery sputum daily (slides).

This shows the lesion in the left lower lung field, somewhat similar to an interlobar effusion; however aspiration was of no avail. This patient refused exploration. He was in fairly good condition early in hospitalization but would not submit to operation. After about two months there was increase in the lesion on the left side and evidence of a fairly typical bronchiogenic spread on the right. His course was down hill and he gradually suffocated. We are glad he refused surgery in view of the present discussion. The gross specimen was much like gray hepatization or the very descriptive term of gelatine infusion. Pus could not be scraped from the surface, however. The photomicrograph shows typical papillary structure. The high power is similar to that previously described.

DR. STEPHENS.—I want to thank the members for this excellent discussion. Of course, we always enjoy Dr. Graham's remarks, and I was happy to hear that he is somewhat intrigued by the possibility that this may be a malignant tumor which has multicentric areas of stimulation due to irritation from a virus. Certainly it has been true in the papillary warts and it may again be true in this disease and opens up interesting channels for future investigation.

DR. O. THERON CLAGETT, Rochester, Minn.—I have a few points to offer which I think are of some interest. We find that this lesion occurs quite frequently; as a matter of fact, 5 per cent of the cases of carcinoma of the lung that have been examined at necropsy in our institution have been of this type. Seven of the ten cases in this group have had metastases to nodes and elsewhere. It is a malignant condition. We have seven cases that have been operated on because they were not definitely recognized as alveolar cell carcinoma preoperatively. We feel as Dr. Graham has indicated, that since it is a multicentric disease, it is not a surgical disease in most cases if its true nature can be recognized preoperatively. These seven cases occurred in the last 189 pulmonary resections for malignancy that we have done. Surgery should probably be avoided if a definite diagnosis can be made. It is of interest, I think, in our experience that this disease occurs six times as frequently in women as in men; the average age has been 51, which is certainly well in the cancer age group.

The most important contribution we have to make is, I think, our sputum studies. It has been pointed out that these patients have a thin watery secretion, and that has also been noted at bronchoscopy. We have a case in which bronchoscopic secretion was taken from each lung and malignant cells were demonstrated in secretions from both lungs. In five of our seven surgical cases Dr. McDonald made a diagnosis of carcinoma of the lung by cytologic examination of sputum; he was unable to determine its type at that time but on reviewing the experience he has with this condition at present he feels that it may be possible to diagnose this particular type of cancer by the cytologic examination of the sputum and bronchial secretions. If this can be accomplished, it will be of considerable benefit in the management of this condition.

DR. EDGAR DAVIS, Washington, D. C.—I believe that pulmonary adenomatosis is found much more frequently than the reports in the literature would indicate. For example, in September, when I reported one of my cases in Miami, there were eight additional cases reported, none of which had been previously reported in the literature. I, personally, feel it is regrettable that our tumor registry in Washington does not have more reports on these cases.

Dr. Stephens is to be congratulated on having a case so closely and definitely related to the illness of sheep. In the last three years, my former associate and I have had three such patients, two men and one woman. The first patient, a woman who had never lived on a farm and had had no contact with a domesticated animal so far as could be ascertained, had bilateral disease with a mass in the right side and right-sided pleural effusion. This fluid was aspirated, and upon examination for carcinoma cells was reported positive. Had we recognized the condition at that time we could have done some interesting investigative work, such as injecting the fluid into mice, guinea pigs, and rabbits, to ascertain whether or not we could transmit the disease to other animals.

The second patient was a man who did live on a farm but who had no sheep nor hogs, so what his source of infection was, no one can say. The third patient, whom I shall report later, was an operative case.

(Slides.) These slides are of a 63-year-old white man who had lived on a dude ranch in Nevada for about six weeks. However, he had not come in contact with any sheep and had not eaten at the ranch, he and his daughter having driven to town for all of their meals. The bronchogram demonstrates a perfectly normal distribution and pattern of the bronchial tree without evidence of bronchiectasis. At bronchoscopic examination I was amazed to see the amount of thin, bubbly, mucoid secretions coming from the left-stem bronchus. The secretions showed what was called carcinoma cells. The next slide is a photograph of the specimen showing multiple adenomatous nodules in the periphery. The next slide is a photomicrograph of the specimen the patient coughed up two days after the bronchoscopic examination. A number of pathologists have examined it and all of them have called it carcinoma.

I have with me a report from Dr. Smetana of the Army Institute of Pathology, that might be both interesting and instructive. He has given me a report on eleven cases; nine of them have already been completely worked up and have been reported by Dr. Swan in an issue

granulation tissue which matures to fibrous tissue, the observation of advanced bronchial destruction makes dense scars and extreme stenoses easily understood. Similar scarring occurs in the lung parenchyma, the pleura, and the pericardium by virtue of the natural healing response to tuberculous, or granulomatous infection. When the architectural weakness of the bronchial wall is added to such pathologic changes, it must be deduced that severe tuberculous tracheo-bronchitis either produces the extensive ulcerations revealed at autopsy, or, when the patient lives, bronchostenosis.



Fig. 1.—Example of contralateral bronchial disease. The extensive disease on the right indicates resection. The left lung is in a good, healed state despite the rather severe bronchostenosis revealed in the planigram.

The presence of stenosis itself does not signify complete healing of a given lesion. However, if, through the bronchoscope, acute ulcerations and inflammations are observed to progress to clean, smooth, stenotic scars, and to remain such, they may be regarded as healed despite the possible persistence of active tubercles in the fibrous scar. Only the upper end of a stenotic bronchus can be viewed. If it appears healed, the remainder of that bronchus is also likely to be healed, especially if the clinical course of the patient and of the lung is favorable, and the sputum is mostly negative. While a stenosis of a given bronchus is usually in a uniform state of healing, the bronchi beyond it may be in any state. This is particularly true of the upper lobe bronchus, and its immediate branches. Active caseation may be found in the upper lobe bronchi, while healed stenosis exists in the main bronchus. In fact, these circumstances should be suspected whenever the sputum is usually positive, the visible bronchial lesion apparently healed, and no open lesion exists in the lung fields.

PLASTIC RECONSTRUCTION OF TUBERCULOUS BRONCHOSTENOSIS WITH DERMAL GRAFTS

PAUL W. GEBAUER, M.D.
HONOLULU, HAWAII

PRESENT day tuberculosis of the trachea and bronchi as seen in living patients alters some of the conceptions of the disease gained from voluminous post-mortem studies of the past. Clinical and autopsy findings indicate a higher incidence in women, a more frequent stenosis of the left bronchus, and a parallelism between involvement of the air passages and advanced, extensive disease of the lungs. Beyond this there is no constant relationship between the location and severity of the tracheobronchial lesion, and the location and extent of the parenchymal disease.

THE STENOSIS

Tuberculous lesions may occur anywhere in the air passages. They do not predominate on the posterior membranous surface. They may involve the circumference of a short segment of a bronchus, or its whole length. Advanced bronchial disease is sometimes encountered in patients with clinically uninvolved lungs. Tuberculous ulceration may be present in the left bronchus while extensive lung disease exists on the right (Fig. 1).

If the pulmonary disease is compatible with life, and especially if it is controlled by treatment, tracheobronchial lesions tend to heal. A small mucosal ulcer resulting from caseation of a submucosal focus may heal without grossly discernable scar. If the elastica is destroyed, healing produces a puckered scar and mild deformity. Breakdown of coalescent caseous foci produces the type of ulceration seen at autopsy and popularized by textbook illustrations. Such extensive lesions in living patients are now rare, and more rarely they heal with the formation of fibrous scars which produce gross deformity of the airway cross section. Whenever inflammation involves the thick, fibrous layer housing the cartilage rings, cicatricial contracture and stenosis always accompany the healing process, because of the natural architecture of the trachea and bronchi. Collapse of the fibrous bronchial wall is caused by dissolution of the supporting cartilages secondary to destructive chondritis. Simple collapse and contraction resulting from loss of supportive framework probably has not been allotted due attention in the pathogenesis of bronchostenosis. Under such circumstances healing of a severe mucosal lesion alone may produce stenosis, without destruction of the fibrous and elastic elements of the bronchial wall, or without the excess production of scar tissue. On the other hand, at operation, caseation of the entire bronchial wall is occasionally encountered, and frequently fragmented or necrotic cartilage is found. If it is borne in mind that tuberculosis heals by the conversion of cellular exudate to fibrous tissue, and by the proliferation of

From the Thoracic Surgical Service, Leahi Hospital.

Read at the Twenty-ninth Annual Meeting of The American Association for Thoracic Surgery, New Orleans, La., March 29-31, 1940.

supply. Regardless, it is a frequent observation, and, in addition, linear incision of a stenotic bronchus usually reveals a more intact mucosa, and less exudate on the membranous portion in contrast to the anterior and lateral walls.

Because of the more favorable pathologic changes, and because of its surgical accessibility, the posterior membranous surface of the trachea and bronchi is considered the most suitable site for surgical attack.

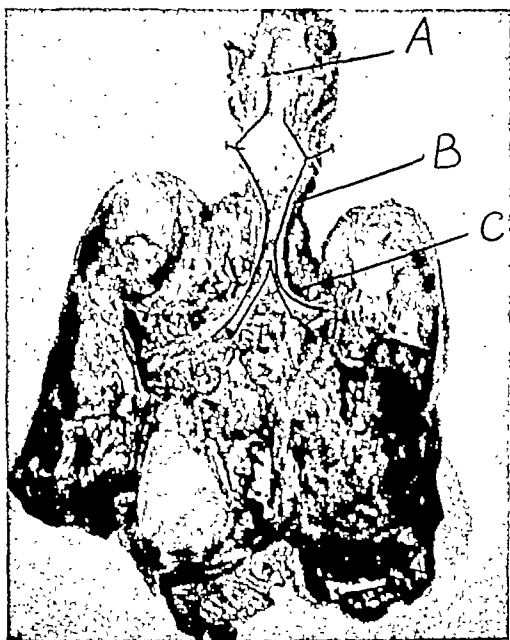


FIG. 3.—Posterior aspect of thoracic contents of patient in Fig. 2. The membranous tracheal wall was almost normal; the paratracheal tissues were particularly clean and free of scar. The hilar structures around the stenosis of the distal right main bronchus were easily separated by dissection. A, Larynx. B, Trachea. C, Right main bronchus.

THE LUNG

If a bronchostenosis is severe enough, or present long enough, functional impairment and subsequent destruction of the respiratory system beyond the stenosis eventually occurs. The pathologic changes distal to a stenosis are dependent on many interrelated factors besides "resistance and virulence." Some of these factors are the extent and course of the parenchymal disease when the stenosis first becomes obstructive, the amount of secondary infection present, the rate of progression, and the degree of obstruction, the effects of treatment, such as lung collapse, and the volume of lung tissue affected by the

As intimated previously, consideration of the effect of dissolution of the supporting cartilages indicates that severe stenosis can occur without preceding extensive destruction of the bronchial wall. More often than not, this concept is supported by operative findings. Frequently the stenosis is a contracture of the bronchus itself. The surrounding lung stroma and mediastinal structures are quite readily separable from the diseased bronchus by surgical dissection. Figs. 2 and 3 are illustrative. Certainly, peribronchial inflammation and scar are not essential for the production of bronchostenosis. Less commonly, extramural scar contributes to the stenosis; sometimes the hilar structures, particularly the pulmonary artery, are encased in hard scar. Such changes follow extensive, deep destruction of the bronchial wall. Fig. 5 is an example. In some instances lymph node perforation of the bronchial wall is a possible etiologic factor.

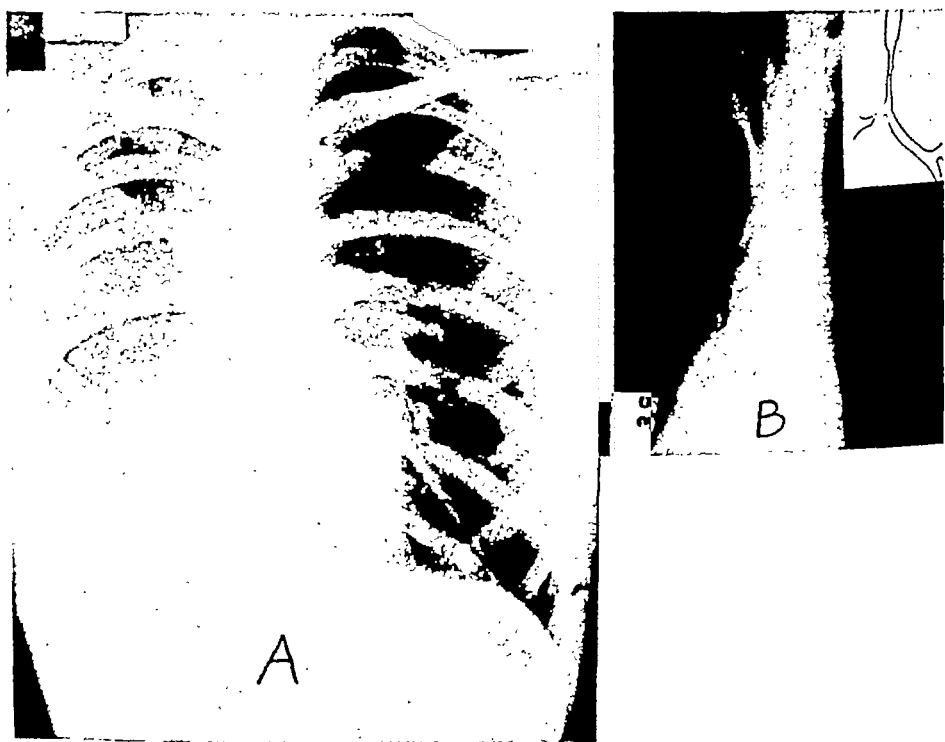


Fig. 2.—Example of severe stenosis resulting from purely intrinsic disease of the air passages. A, Atelectasis and destruction right middle and lower lobes, some infiltration in the upper lobe; left lung is clear. B, Extreme stenosis of the thoracic trachea, resulting from extensive ulcerocaseous tuberculosis. Healing was observed bronchoscopically during and after treatment with streptomycin.

The posterior membranous portion of the larger bronchi contains no cartilage. Healed lesions here are narrow and contracted, but not to the same extent as the remainder of the bronchial circumference. The element of wall collapse is lacking, and the resultant difference in the disposition of scar tissue found at operation is sometimes striking. The same disparity does not occur in surgical specimens exhibiting tuberculous stenosis of small branch bronchi, where cartilage plates are irregularly disposed about the whole circumference. The inequality in the degree of scar may not be due to the absence of cartilages posteriorly. It may be due to a difference in healing properties, or in blood

In the beginning, episodes of atelectasis are intermittent, and frequently influenced by attempts at collapse. Some lung segments may be spared; frequently lung segments and lobes quite free of disease are "shut off" by the stenosis. Later on, atelectasis either becomes persistent with its usual consequences, or one of them, such as bronchiectasis, dominates the picture (Fig. 6). Up to this point resection of the involved lung and its stenotic bronchus is clearly indicated.



FIG. 5.—C, Patient of FIG. 4, after left pneumonectomy; also an example of extramural tissues contributing to stenosis; no "flare-up," right base. D, Planigram before operation showing stenosis of left bronchus and lower trachea. Immediately after removal of the left lung and its hilar scar, it was possible to advance a 32 French intratracheal catheter into the right bronchus. Bronchoscopy before operation had revealed a 6 mm. lumen in the lower trachea. E, Planigram after operation demonstrates the increase in tracheal lumen after resection of extramural scar.

Now and then healed stenosis of the main bronchus is accompanied by parenchymal disease, static or progressive, which remains confined to the upper lobe while the remaining lobes are free of disease, and unaffected by the main bronchial lesion. Such circumstances are not uncommon; however, they do not exist forever, and sooner or later the good lobes succumb to the stenosis through which they must function. If patients can be detected clinically before this, resection of the diseased lobe and reconstruction of the airway to the good lobe is considered more rational treatment than excision of everything distal to the stenosis. Cases 1 and 2, cited later in this report, belong to this group.

The third group of patients have healed bronchostenosis with functional or organic occlusion. The component lung is atelectatic and usually consists of

stenosis. None of the changes resulting from stenosis are beneficial, and the usual elements of the combine which eventually destroys the distal lung tissue are atelectasis, fibrosis, progressive tuberculosis, bronchiectasis, nonspecific pneumonitis, and abscess formation. Any one process or any combination may dominate the clinical picture, which is as variable as the responsible processes. However, these variations are more those of degree than of kind, for in general all the pathologic changes are destructive and progressive. The progression may be slow or rapid. Regardless of the mechanism of destruction, lung function is lost and illness is the patients' lot. Among the various clinical states produced by such illness, the following types are clinically prominent.

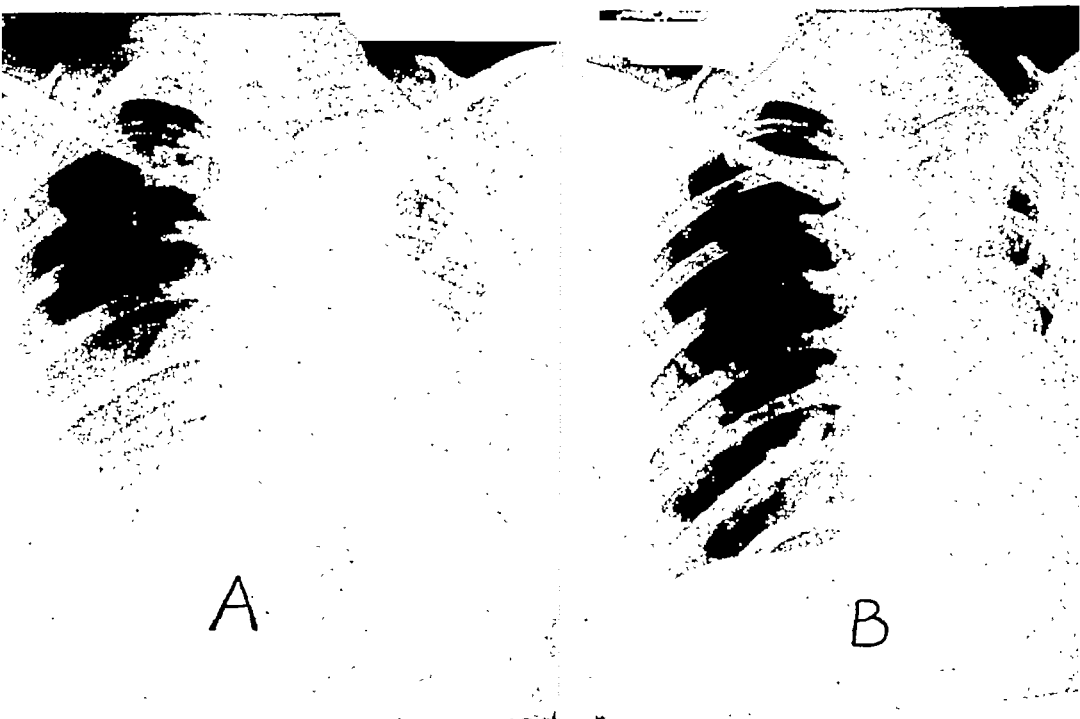


Fig. 4.—Example of Group 1, representing extension of parenchymal disease during active phase of bronchial lesion. A, Left lung destroyed; acute "spread," and fluid base of right lung. B, Clearing following streptomycin.

The first group of patients have rapidly progressive tuberculosis. The lung involvement is usually extensive and advances in step with the stenosing process in the bronchus. By the time the bronchial lesion is converted from an ulcerocaseous phase to stenosis, the lung disease is far advanced. The only hope is to bring the patient to a point where excision of the bronchus and its constituent lung can be done. The patient presented in Figs. 4 and 5 is an example. Streptomycin cleared the right lung and brought the tracheobronchial lesion to a surgically receptive state.

The second group of patients have healed or mildly active bronchial lesions. Their lung disease may be little or advanced; in either case, it is progressive and contraindicates any attempt to relieve the stenosis surgically. Sometimes the progression is rapid, especially during, or following a period of atelectasis.

healing is not complete, during which they actually belong in the second, or previous group. It is inadvisable to wait for ultimate, natural healing in stenosis of a lobar bronchus, because of its rarity. If the bronchus is occluded the component lung is useless and chances are it will not remain innocuous. Lobectomy should be done, providing the remainder of the lung on the same side is clinically "healed" or free of disease. Certainly, natural healing should not be awaited hopefully in main bronchial lesions. If the lung is diseased it should be removed; if it is in a good healed state, reconstruction of the stenotic bronchus seems clearly indicated.

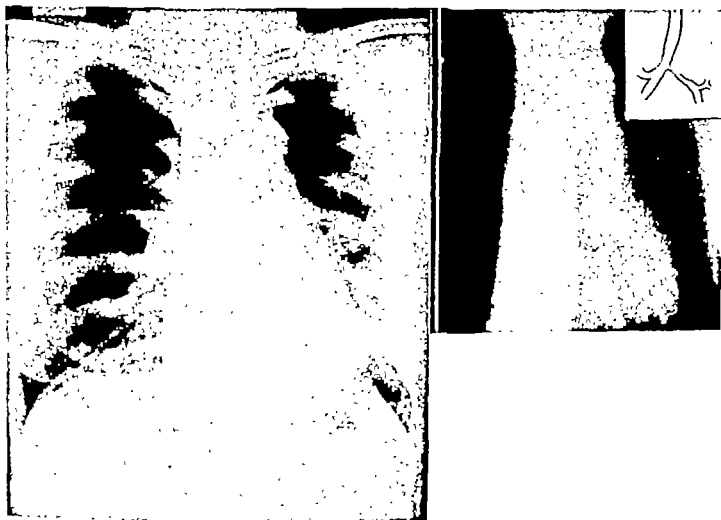


Fig. 7.—Example of Group 4. The left lung is healed with some fibrosis. The stenotic left bronchus had a 3 mm. lumen at the carina, not enlarged by repeated dilatations. The patient has negative sputum, a persistent wheeze, intermittent cough, and diminished breath sounds. Reconstruction of the left bronchus, rather than pneumonectomy, seems indicated.

The characteristics which make the fourth group of patients clinically prominent are healed, quiescent, or minimal lung disease, and a healed, stenotic bronchus. A minimal airway exists permitting lung aeration. Over a period of months or years, episodes of sputum block, intractable cough, atelectasis, and dyspnea occur with irregular frequency. Except for cough and wheeze, the true disability is periodic, but every bout of atelectasis, and every "cold" hazards a "setback," "spread," or further lung damage. The ultimate prognosis for a given lung is doubtful. Some degree of functional impairment is always present, and the passage of time leads to increasing impairment secondary to fibrosis, atelectasis, bronchiectasis, and so forth. In many of these patients tuberculosis produces a scar in the bronchus and lung, and then drops out of the picture. The offending agent is a structural deformity of the air passage invoking a chronic, recurring disability for which most patients willingly

a small fibrotic mass containing "burned out," or healed disease, or encapsulated caseous foci. The patients have negative sputa and are clinically well. Such conditions represent the ultimate in natural healing and are common in small segments of lung. An entire lobe may heal in this fashion, but rarely does a whole lung. When a large or a main bronchus is occluded, some degree of

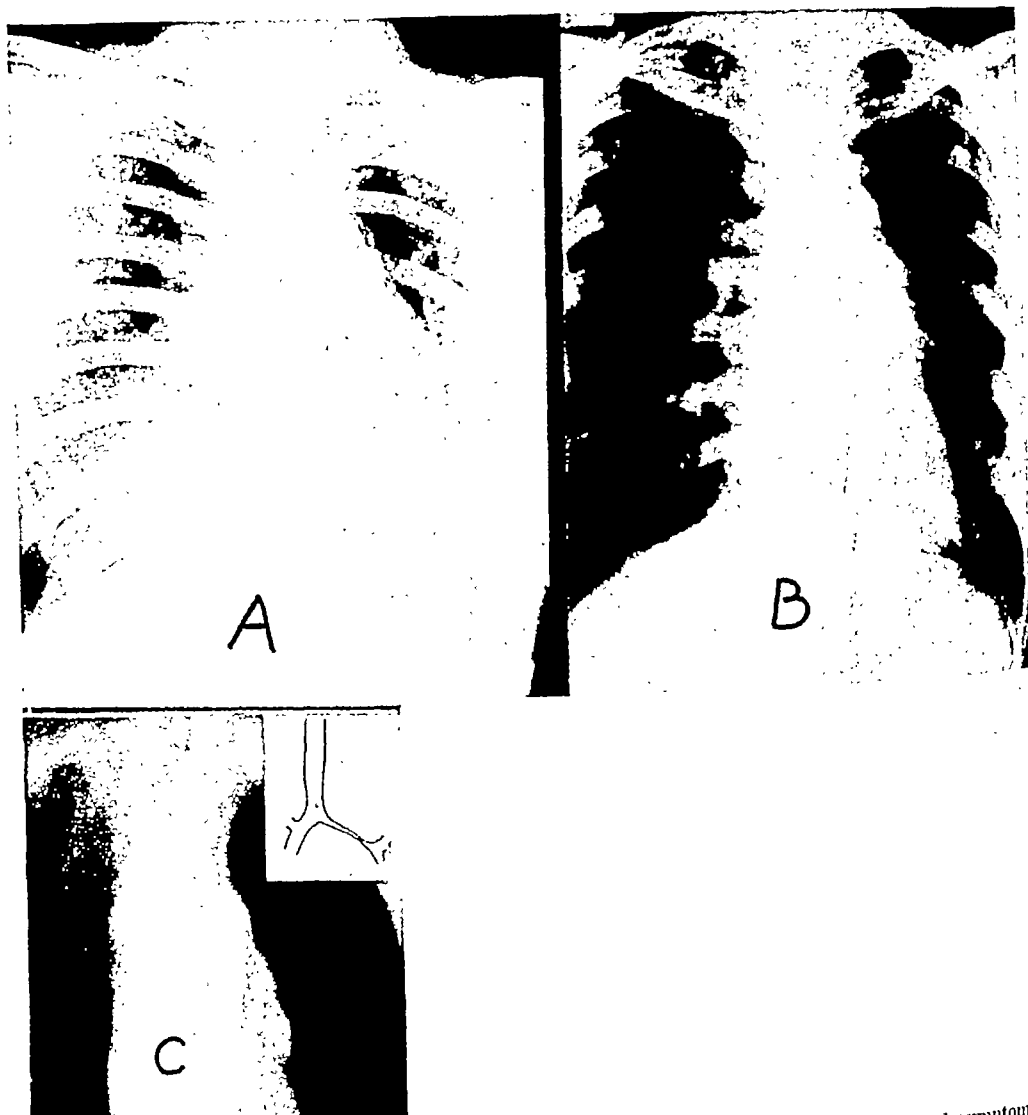


Fig. 6.—Example of Group 2. A, Episode of left lower atelectasis with marked symptoms. B, Spontaneous clearing four months later. C, Planigram illustrates left bronchostenosis. The left lung cleared further with intensive antibiotic therapy, but pneumonectomy was indicated because of irreversible bronchiectasis of both lobes, and unhealed tuberculosis in the upper lobe. The stenotic bronchus was well healed and could have been reconstructed. This was contraindicated by the previous findings, which were established by complete bronchotomy, as well as by subsequent examination of the surgical specimen.

infection usually supervenes and the chain of events leading to lung destruction begins. Even with insignificant infection bronchiectasis occurs. Case 4, cited later, belongs in this group.

Most patients in the third group have fairly good resistance and have done their share of "curing." Nevertheless, they all pass through a stage when

primary healing of an amputated bronchus can always be expected if it is handled carefully, sutured accurately, its blood supply preserved, and infection avoided or controlled. Uncomplicated bronchotomy and suture, despite the presence of some infection, has been reported in man.^{4,5} Belsey⁶ briefly reported an instance of tracheal reconstruction with a fascia-wrapped spiral of stainless steel wire. Gibbon⁷ mentioned the filling of a tracheobronchial defect with a segment of costal cartilage wrapped in a pedicle of parietal pleura. Finally, excisional lung surgery has shown that the healing of a bronchial stump is enhanced by the formation of scar arising from the adjacent lung, pleura, or mediastinal tissues. In fact, sometimes a large portion of the scar of a healed bronchial stump grossly appears to have been contributed by these structures, or by the organization of blood clot which they support. Such findings suggest that the mediastinal bed of the air passages has good sealing and healing properties of its own. On the basis of experimental observations and clinical experience, it seems logical to expect plastic procedures and grafts of the bronchi to be feasible and practical.

Any substance used in bronchial grafts should meet certain essential requirements. First, it should provide an adequate, air-tight passage. Second, it should afford a semirigid, noncollapsible support. Third, it should serve as a scaffold for the regeneration of epithelium. Fourth, and finally, an ideal graft should survive as living, transplanted tissue, and, at the same time, provide wall structure. This final requirement is not considered completely essential for a good ultimate result. Failing to "take" in entirety, a graft could still function as a foundation for natural healing and regeneration. It could provide a framework for such processes during its disintegration and replacement. Fascia and Gelfoam apparently behave in some such fashion in the experimental animal. Actually, it is possible that metaplasia, and even metamorphosis, might occur in any type of grafted tissue, so that ultimately it would simulate the neighboring bronchial structures.

The dermal layer of the skin probably fulfills the preceding requirements better than any other living tissue. Its properties, and the properties of whole skin used in place of fascia, or buried to fill defects, are recorded in medical literature. May and Spann⁸ have summarized the early use of the dermal graft. In personal experience, it has been found useful as a heavy ligature for partial occlusion of the thoracic aorta above an aortic aneurysm, or as a bronchial plug, anchored with wire sutures, into the bronchial fistulas of exteriorized lung cavities. A rapid "take," and the growth of epithelium over the dermal plugs, has been perceived in such instances. The dermal skin is thick, tough, and elastic. It is rich in vascular and lymphatic channels. It is naturally designed for the foundation of a surface. It holds sutures tenaciously; it does not separate or tear when pierced with a needle, as does fascia, pleura, or epidermis. For these reasons, and because of its thickness, it is well adapted for airtight suture lines. When applied to a bronchus as an inlay graft, the cut surface of the deep epidermis and the skin papillae are presented to the bronchial lumen. This surface forms a suitable bed for the ingrowth of bronchial epithelium. Should this fail to occur, new squamous epithelium can regenerate from the papillae, thereby affording a good opportunity for metaplasia. If the epidermis

submit to excisional surgery. This relieves their symptoms. Also, it frequently relieves them of considerable volumes of good lung tissue, and that is the trouble. Pneumonectomy or lobectomy should be performed forthwith when the lung or a lobe is diseased, but one hesitates advising removal of a good lung because its air passage is stenosed, even with the suspicion that matters will get worse as time goes on. Figs. 7 and 8 illustrate two patients in this group.

The preceding rather gross evaluation of the clinical circumstances ordinarily obtained from healed or healing tuberculous bronchostenosis designates a number of clinical states in which reconstructive surgery of the air passages seem highly desirable.

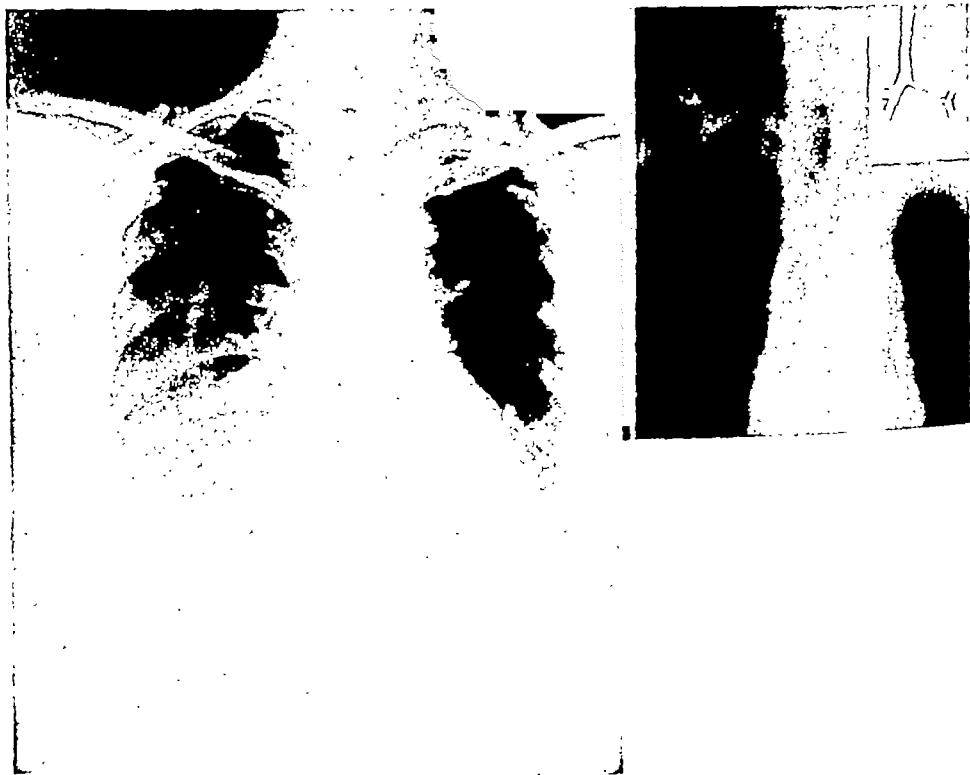


Fig. 8.—Another example of Group 4. Scattered, healed disease, possibly some bronchiectasis, exists on the left. The planigram shows stenosis of the left bronchus. Symptoms are a rather severe, persistent cough, wheeze, and episodes of sputum block. Sputum has been negative for over one year. Reconstruction of the left bronchus is indicated.

PROPOSAL

The surprising healing and regenerative powers of the epithelium and other wall structures of the dog's trachea and bronchi have been demonstrated by Taffel,¹ Hanlon,² and Daniel.³ In the experimental laboratory, the growth of epithelium over degenerating fascia, gelatin sponge, and totally foreign substances, as well as the regeneration of cartilage rings about such structures as glass tubes, have been observed, indicating a healing response of these tissues beyond ordinary expectation.

In addition to these favorably prophetic experimental findings, certain clinical facts suggest that the human trachea and major bronchi have similar properties, and might be highly receptive to reconstructive surgery. Clean,

can be more narrow and less curved. The length of the bronchial incision is measured and a pattern cut from paper, which is curved and approximated to the bronchial defect. The graft is cut somewhat larger on all sides. The wire

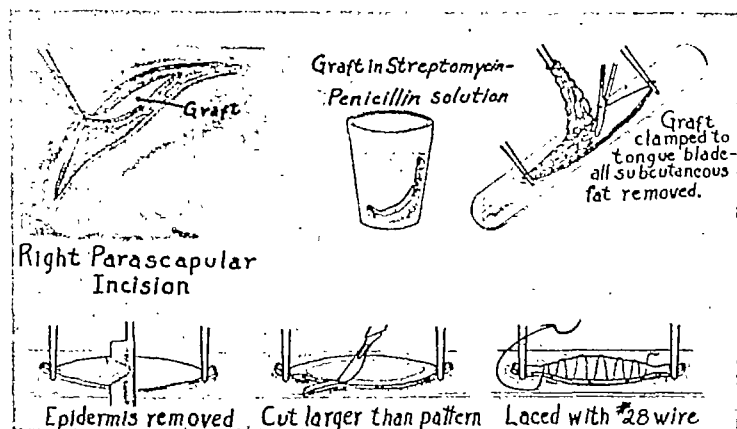


Fig. 9.—Drawings illustrating the technique of preparing a dermal graft for a bronchus.

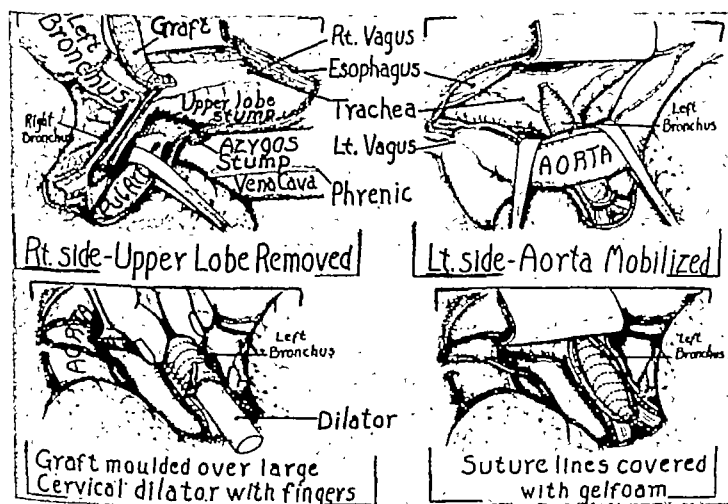


Fig. 10.—Drawings illustrating the exposure of the main bronchus and lower trachea on the right and left sides, and the technique of inlaying dermal grafts.

were not removed, it would probably slough off and be expectorated. Until then, a suitable surface for the ingrowth of bronchial epithelium would be absent. For the above reasons the derma is recommended for bronchial grafts, and is considered superior to full thickness skin.

TECHNIQUE

Dermal skin fulfills all the proposed requirements except semirigidity. This can be provided by stainless steel wire when it is laced through the graft, as depicted in Fig. 9. This disposition will not prevent give and motion in either axis, so that some degree of pliability is preserved. The technique of dermal grafts to the main bronchi is illustrated in Figs. 9 and 10. Experience to date has uncovered some details that might be useful to those interested in performing the procedure.

The face down, prone position, with the operated side tilted lowermost, is unquestionably the best. It permits a direct posterior exposure, reduces mediastinal motion, and prevents "spill" to the good side.

Exposure on the right is simple. On the left it is usually necessary to mobilize the aortic arch for adequate exposure of the lower trachea and bifurcation. Such mobilization is likely to injure the recurrent laryngeal nerve unless it is identified and protected.

The esophagus tends to bulge into the field and must be retracted; this frequently exposes the thoracic duct which can be protected with a pack. Frequently, the pulmonary artery is involved in an upper lobe bronchial scar, and separation injures it. It must be preserved. This has been accomplished by temporary occlusion with a soft clamp, and the application of a small patch of Gelfoam held in place with overlying arterial sutures taken in the adjacent arterial wall.

Before incision of the stenotic bronchus, the intratracheal catheter is advanced by the anesthetist, and guided into the opposite main bronchus with a palpating finger. After the bronchus is opened, it is carefully inspected, washed with streptomycin-penicillin solution, and packed with gauze soaked in the same solution. In the meantime a suitable dermal graft is fashioned from the ellipse of skin removed at the primary incision.

The graft should be cut somewhat larger than would be expected. It does not have to be stretched taut. It should come to abrupt ends. The loops of wire (28 gauge) should be wide enough to provide a fairly round contour to the graft. This is most important, for insertion of a flat graft will produce a wide slit, rather than an open lumen. The width of the graft, therefore, is determined largely by the width of the wire loops. The elastic derma should be about the same width, but does not have to be wider. A graft $\frac{3}{4}$ inch wide will form a $\frac{1}{2}$ inch lumen diameter when it is curved into a semicircle. The size of the graft is a matter of judgment as well as measurement. It depends on the width of the opened bronchus, and its shape. If the posterior bronchial wall is thin and retracts, leaving a flat scar, the graft will have to be wide with a full curve. If the bronchus itself has some curve and is fairly wide, the graft

the bronchial lesion followed treatment with streptomycin. Thereafter, the course was one of intermittent atelectasis with positive sputum, cough, wheeze, and diminished breath sounds, despite a second course of streptomycin. Four months previously, bronchoscopy revealed a healed stenosis of the right bronchus at the carina, with a 3 mm. lumen. Planigrams showed this lesion to involve the whole right bronchus. Sputum culture was still sensitive to streptomycin.

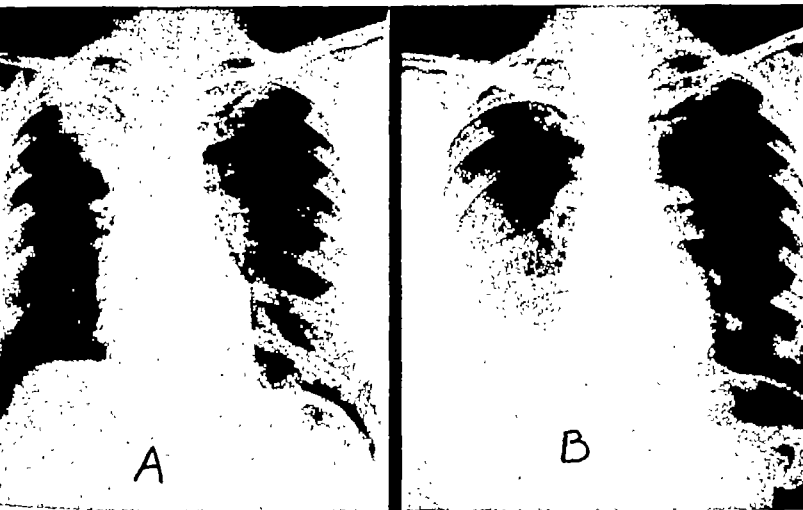


Fig. 11 (Case 1, A. F.).—A, Before operation; recently increased partial atelectasis right upper lobe. Sputum positive. B, After right upper lobectomy and dermal graft of right main bronchus. Sputum negative.

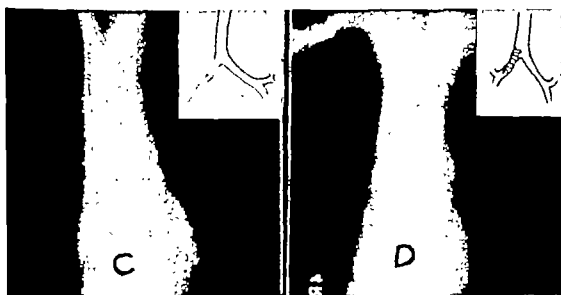


Fig. 12 (Case 1, A. F.).—C, Planigram before operation; irregular marked stenosis right bronchus. D, Planigram two months after right upper lobectomy and graft of main bronchus. The wire within the graft is poorly reproduced, and the first portion of the main bronchial lumen is in a more anterior plane of focus. Bronchoscopy; graft covered with epithelium, lumen oval and distorted when forced anterior by a straight scope. Bronchoscope with an external diameter of 8 mm. passed into lower lobe without difficulty. Graft possibly too small and not sufficiently rounded.

is laced through the graft for a distance approximating the bronchial incision. Long loops should oppose the narrow portions of the bronchus. The short loops, on the ends, oppose the trachea and bronchi above and below the stenosis.

Redundant skin is left at each end of the graft, by which it is manipulated. The cut surface of the epidermis is applied to face the bronchial lumen. The graft is anchored to the posterior tracheal surface, proximal to the bronchial incision, with a mattress suture. Here the graft lies on the posterior tracheal wall; distal to this the graft is brought into the defect as an inlay, and sutured into place. Every now and then a suture is taken to include a wire loop. The distal end of the graft is fixed in a similar fashion. Thus a small wedge of graft is not made to fit into the angles of the bronchial incision, the most likely point of leakage, or graft slough. Stainless steel, 35 gauge, multistrand, wire sutures on atraumatic needles are used throughout.

Before completing the sutures a large cervical dilator is inserted into the bronchus, and the wire loops and graft are molded over it with the thumb and forefinger (Fig. 10). This produces a round contour, and forms an open bronchial lumen in contrast to a slit. The suture lines are then covered with narrow strips of Gelfoam previously soaked in streptomycin-penicillin solution. These strips should cover only the suture line and not the main surface of the graft. It is hoped that the latter will be a source of blood supply. The intratracheal catheter is then drawn back into the trachea, the lung or lobe inflated and deflated, and the graft tested for air leaks.

Postoperative treatment is the same as for any resection, plus the routine use of antibiotic inhalations and expectorants. Bronchoscopy is done if signs or symptoms of bronchial obstruction occur. It is not warranted for simply "a look at the graft." As a rule, a good airway exists immediately after operation. Obstruction by granulation tissue, loosened mucosa, and organized exudate is likely to occur. When it produces a wheeze it should be removed by bronchoscopic aspiration.

The following four cases are presented only as evidence of the immediate efficacy of dermal bronchial grafts. All the patients are too recently operated upon to warrant any statements regarding the final fate of such grafts, or long-term end results. In each case, by present standards, pneumonectomy was indicated and avoided. So far, each clinical result has been good, and all the patients are well. On the other hand, postoperative bronchoscopic findings have been difficult to appraise and, on the whole, disappointing. Despite apparent full "takes," and early epithelization, the bronchial lumen attained at operation, has not persisted unchanged. This is believed to be the consequence of imperfect technique and inexperience, or poor timing of the procedure in respect to the state of bronchial healing. It could mean, however, that stenosis will eventually recur.

CASE REPORTS

CASE 1.—A. F., woman aged 29 years (Figs. 11 and 12). Minimal pulmonary tuberculosis was discovered three years previously. One year previously, a sudden, acute illness was characterized by a positive sputum and a cavity in the right upper lobe. Right pneumothorax was abandoned after it produced a wheeze and atelectasis of this lobe. Bronchoscopy disclosed ulcerocaseous involvement of the right main bronchial orifice. Good healing of

The patient's temperature rose to 102° F. on the first day. It was 100° F. on the third day and normal thereafter. A pulse of 120 on the first day was normal after the fifth. The lower lobe filled the pleural space by x-ray examination on the first day. Normal breath sounds were audible on the left at all times. The right pneumothorax was maintained.

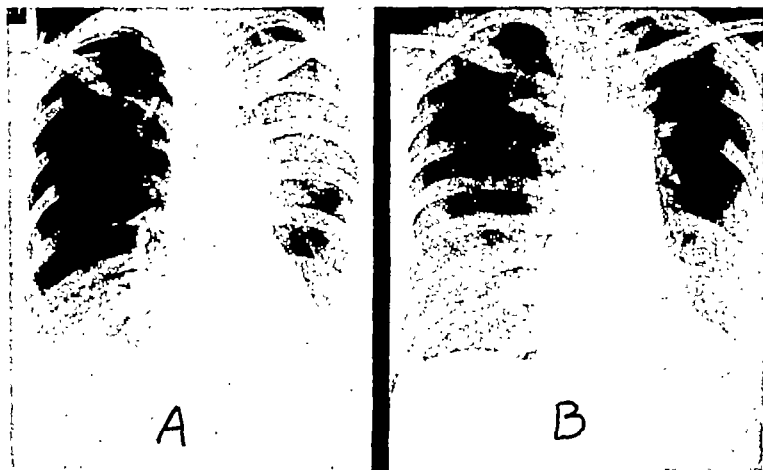


FIG. 13 (Case 2, D. K.).—A, Before operation; apical pneumothorax, right, atelectasis left upper lobe. Sputum positive. B, After left upper lobectomy and dermal graft to left main bronchus. Sputum negative. Right pneumothorax maintained.

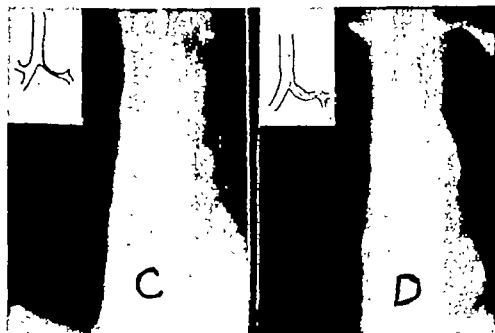


FIG. 14 (Case 2, D. K.).—C, Planigram before operation; moderate stenosis whole left bronchus, probable occlusion upper lobe bronchus. D, Planigram after operation; good bronchial lumen. Bronchoscopy; scope with an external diameter of 10 mm. passed beyond graft to the lower lobe. Lumen somewhat flattened; graft would be better with a more round contour.

A slight wheeze developed after three weeks and bronchoscopy revealed some fibrinous exudate at the upper angle of the graft which bulged into the tracheal lumen. The graft was clean and pink, except for a few small pale areas. A standard 7 mm. bronchoscope, which has

At operation the middle and lower lobes were blocked in an inflated state. The upper lobe was hard and could not be inflated; its bronchus was scarred, stenotic, and occluded with caseous exudate. The upper lobe was removed. The right main bronchus was markedly narrowed. The posterior surface was easily exposed, narrow, and slightly thickened. Palpable and visible cartilage fragments were few in number and irregularly disposed. The posterior wall was opened from the lower lobe into the trachea. The lower and middle lobe bronchi were normal. Clean mucosa existed on the posterior wall but the anterior bronchial wall was caked with a moist, yellowish-white, fibrinous membrane which was removed, revealing a clean, finely granular, vascular surface. The stenosis was most marked at the carina and opposite the upper lobe. A graft was inserted, as depicted in Figs. 9 and 10. It was not well rounded. A lumen was obtained which admitted an aspirating tip measuring 9 mm. in diameter. Inflation and deflation of the middle and lower lobes was easily accomplished.

The highest postoperative temperature was 100.4° F. on the first and second days. The pulse did not exceed 100 and was normal after the fourth day. Expansion and aeration of the right middle and lower lobes have been present by physical and x-ray examination ever since operation. Because of a wheeze which increased in pitch, bronchoscopy was done on the eighteenth day. The graft was clean, pink, and appeared covered with epithelium. The bronchial lumen was oval in shape and partially obstructed by thin, edematous, filmy tissue, or exudate, hanging like a curtain from the anterior bronchial wall (site of the caked exudate at operation). It was easily removed by aspiration, obtaining a bronchial lumen which admitted a scope (external diameter 8 mm.) into the lower lobe. The wheeze disappeared and later recurred. Bronchoscopy six weeks after operation again showed a small amount of tissue on the anterior wall at a lower level. It was removed by suction. The stump of the upper lobe was healed and clean. The last bronchoscopy, nine weeks after operation, showed complete epithelization of the graft. The most proximal wire loop of the graft bulged into the trachea. Just above the middle lobe orifice, at the distal end of the graft, the bronchial lumen appears as a slit. It accepts a 5 mm. bougie easily. This graft is regarded as too short, too flat, and too narrow near the ends.

CASE 2.—D. K., woman aged 38 years (Figs. 13 and 14). Diagnosis pulmonary tuberculosis made four years previously because of light bilateral apical infiltration. Wheeze and increase in extent of disease one year previously was treated with pneumoperitoneum and streptomycin. The wheeze was relieved but the sputum remained positive. Pneumoperitoneum was abandoned and right pneumothorax started nine months previously. Two months before operation the left upper lobe became atelectatic with the usual signs and symptoms of bronchial obstruction. Bronchoscopy revealed a healed stenosis of the left main bronchus with a 3 mm. lumen at the carina. Within two months an appearance suggesting bronchiectasis of the left upper lobe occurred in the roentgenograms. Planigrams showed narrowing of the whole left bronchus, normal lower lobe, and probable occlusion of the upper lobe. The sputum was positive.

At operation the lower lobe was normal. The upper lobe contained only small patches of air and could be partially inflated from the lower lobe across the hilar portion of the fissure. The apex was hard and irregular; the lingula was solid. The upper lobe bronchus was very small and inseparable from the pulmonary artery. Careful dissection broke into both structures at once. Caseation of the bronchial wall was complete at this point. The artery was temporarily occluded and repaired with a small patch of Gelfoam held in place with overlying arterial sutures taken in the adjacent adventitia. Dissection of the upper lobe bronchus centrally provided solid scar for its suture. The upper lobe was removed. The entire left bronchus was opened. There was a stenosis of varying degree. The lumen at the carina was about 3 mm. The largest lumen elsewhere was 5 mm. The posterior membranous portion was somewhat thickened and narrowed; it was bordered by many fragments of intact cartilage. There was no exudate in the lumen. The mucosa was thin and apparently absent in some areas where the surface was uneven and finely granular. The lower lobe bronchi were normal. A large graft was applied, as depicted in Figs. 9 and 10, and the lower lobe easily expanded.

effusion. Two years before operation, reactivation occurred and bronchoscopy disclosed ulceronecrotic involvement of the left upper lobe bronchus. This lesion regressed and flared intermittently for over a year. Healing finally followed a three-month course of streptomycin. Bronchoscopy showed contraction of the left main bronchus but the upper lobe orifice could not be seen. A cavity persisted in the upper lobe and a first stage thoracoplasty was done. Dyspnea was marked in the early postoperative period. After a few months even mild exertion produced some shortness of breath. Ventilatory studies contraindicated further thoracoplasty. The sputum remained positive. Planigrams showed sharp angulation and stenosis of the distal left bronchus, and a thick-walled cavity in the partially collapsed upper lobe. The extremely low respiratory function was out of proportion to the x-ray and clinical findings. It was felt that dyspnea was possibly due to expiratory block of the left lower lobe. Despite the obviously desperate risk, operation was undertaken with the hope of excising the upper lobe and restoring the lower lobe to better function by correction of the bronchial deformity.

At operation the hard, airless upper lobe was freed in the extrapleural plane. The fissure was fused. The lower lobe contained no palpable induration; it did not deflate except with manual pressure. The upper lobe bronchus was represented by dense scar inseparable from the pulmonary artery. A portion of the bronchial wall was excised and left adherent to the artery. The upper lobe bronchus contained caseous exudate. The upper lobe was removed. The whole main bronchus was scarred. Cartilages were absent in the distal portion, opposite the upper lobe stump, and here the bronchus was soft and collapsible. The lumen measured about 4 mm. The bronchus made a sharp angle at this point and was lined with thin mucosa which appeared normal on the posterior wall. The lower lobe bronchi were normal. The distal half of the main bronchus was reconstructed with a dermal graft. Following this, the lower lobe could be inflated at will. The left phrenic nerve was inadvertently crushed with forceps during closure of the mediastinal pleura.

On the first postoperative day the temperature was 102° F. and pulse 140. On the third day the temperature was 100° F. and pulse 110. The left lower lobe was well expanded but aerated poorly. Fluoroscopy revealed elevation and paralysis of the left diaphragm. On the sixth day breath sounds were more diminished and bronchoscopy disclosed partial bronchial obstruction by a flap of loosened mucosa just above the graft. The graft was intact and pink in color. However, the bronchial wall was rather soft and the lumen flattened. It was possible to pass a scope, with an external diameter of 8 mm., beyond the graft. Following bronchoscopy, aeration of the left lower lobe improved and three weeks later the chart curves were normal. Since then there has been no indication for bronchoscopy but the left diaphragm has not recovered its function.

CASE 4.—R. C., woman aged 26 years (Figs. 17 and 18). Onset two years previously with acute laryngitis and a dense left upper lobe containing a cavity. The larynx improved and bronchoscopy a year later showed active caseous involvement and stenosis of the left bronchus at the carina. Three months later, after streptomycin, this was healed and the lumen measured 2 to 3 mm. The sputum became negative. Atelectasis of the left upper lobe was intermittent until one month before operation, when it increased and persisted. There was increased cough and occasionally purulent sputum which was negative. Examination revealed a wheeze, and diminished to absent breath sounds over the whole left chest. Planigrams showed stenosis of the whole left main bronchus and apparent occlusion of the upper lobe bronchus.

At operation the lower lobe was hyperexpanded. It contained no palpable nodules or induration. The upper lobe was a small, firmly puckered structure drawn down toward the hilum. It contained many small, hard, palpable nodules. The secondary bronchi of the upper lobe were all dilated. The upper lobe bronchus itself was short, contracted and hard. The lumen was completely occluded by healed scar. The upper lobe was removed. The main bronchus was markedly contracted. The posterior surface was easily exposed. The membranous portion was narrow and only slightly thickened. Irregular fragments of cartilage were visible and palpable at the margins. The lumen was lined with thin mucosa and exhibited numerous pits and linear depressions. There was no exudate. The lower lobe

an external diameter of 9 mm., was passed into the lower lobe. The bronchial lumen was somewhat flattened; it is felt that a more rounded graft would have been better. There has been no further indication for bronchoscopy and the sputum is negative. The right pneumothorax is being discontinued.

CASE 3.—E. Y., woman aged 24 years (Figs. 15 and 16). Left pneumothorax, started for an upper lobe cavity in 1942, was discontinued after two years because of pleural

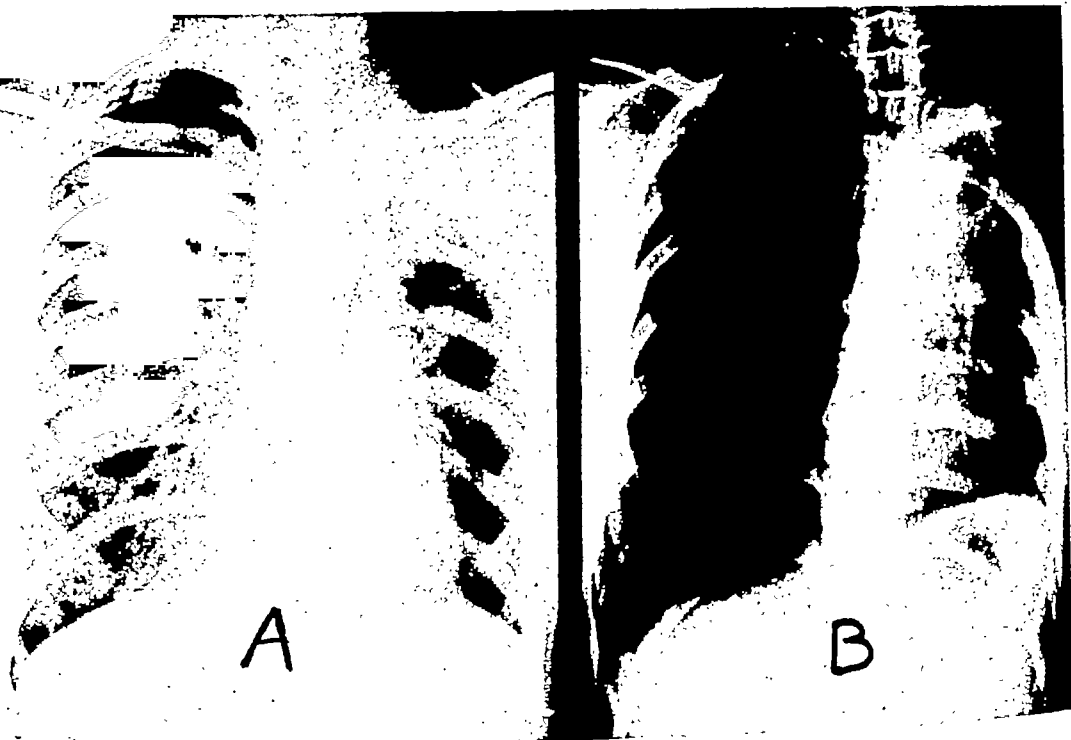


Fig. 15 (Case 3, E. Y.).—A, Before operation; normal right lung, incomplete thoracoplasty left. Ventilatory studies much poorer than indicated by roentgenogram. Second stage thoracoplasty avoided because of dyspnea with mild exertion. Sputum positive. B, After left upper lobectomy and dermal graft to the distal half of the left bronchus. Paretic left diaphragm from operative trauma is responsible for diminished aeration of the left lower lobe. Despite this, its clinical function is as good, if not better, than before lobectomy. Sputum negative.

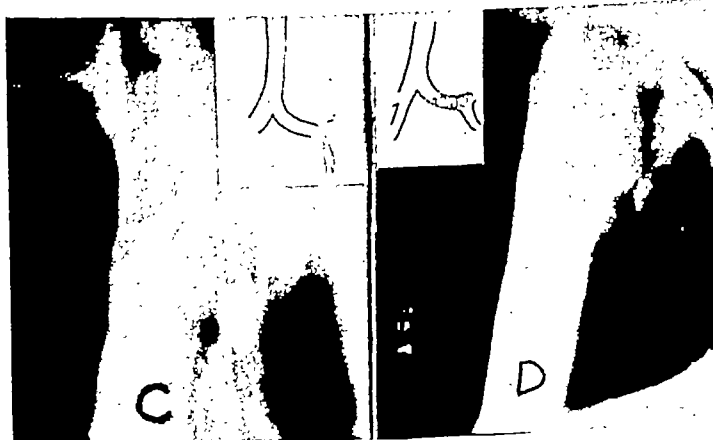


Fig. 16 (Case 3, E. Y.).—C, Planigram before operation; stenosis distal portion left main bronchus with marked angulation, and severe stenosis upper lobe bronchus which possibly leads to a slit cavity. Probable functional block of lower lobe in expiration. D, Planigram after operation; bronchial lumen good, angulation corrected. Bronchoscopy; scope with an external diameter of 8 mm. passed into lower lobe. Whole bronchial wall is soft and the lumen varies in shape as the bronchus is straightened by the scope. A larger graft would be more satisfactory.

first day. The drainage tube and suction were discontinued after forty-eight hours. Since operation, normal breath sounds have been audible over the left chest. Mild dysphagia was present for two weeks. The left vocal cord was still paralyzed five weeks after operation.

At bronchoscopy the graft appeared covered with epithelium. The upper end of the wire within the graft projected from the posterior tracheal wall. The lumen at the carina was about 5 mm. This narrowing was due to a crescent-shaped partition of tissue which was displaced with the tip of the scope. A standard 7 by 40 scope was passed to the lower lobe branches. Beyond the carina the bronchial lumen was clean and large.

This was a big, well-rounded graft. The recurrent narrowing at the carina was disappointing in view of the excellent state of the graft and the bronchial lumen beyond it.

REFERENCES

1. Taffel, M.: *The Repair of Tracheal and Bronchial Defects With Free Fascia Grafts*, Surgery 8: 56, 1940.
2. Hanlon, C. R.: *Observations on the Use of Gelatin Sponge in Closure of Experimentally Produced Defects of the Bronchus*, Surg., Gynec. & Obst. 86: 551, 1948.
3. Daniel, R. A.: *The Regeneration of Defects of the Trachea and Bronchi. An Experimental Study*, J. THORACIC SURG. 17: 335, 1948.
4. Goldman, A.: *The Surgical Treatment of Bronchial Adenoma*, Dis. of Chest 13: 321, 1947.
5. Langston, H. T.: *The Indication for Posterior Transpleural Bronchotomy in the Management of Intra-bronchial Tumors*, Surg., Gynec. & Obst. 86: 192, 1948.
6. Belsey, R.: *Stainless Steel Wire Suture Technique*, Thorax 1: 39, 1946.
7. Gibbon, J. H.: *Discussion of paper by Daniel*.
8. May, H., and Spann, R. G.: *Cutis Grafts for Repair of Incisional and Recurrent Hernias*, S. Clin. North America, 28: 517, 1948.

DISCUSSION

DR. E. J. O'BRIEN, Detroit.—When I was in Hawaii in January, I was so impressed with Dr. Gebauer's results with dermal graft that I asked him to present his work at this meeting. With his inherent modesty he was reluctant to report so small a series of successful operations, but I persuaded him to come and present his work as a preliminary report.

Dogs are very difficult to obtain in Hawaii. I, therefore, promised him I would try some cartilaginous transplants, portions of bronchi, tracheas, etc.

Dr. Jackson, who is one of our residents, has done much of this work. With your kind permission I should like to have him tell you what we have found to date.

I want to make it clear that this work was done at the instigation of Dr. Gebauer, and as such is really a collaboration and not a discussion.

DR. T. L. JACKSON, Detroit.—We have been interested in the healing of the trachea and bronchus for some time. At present we have ten dogs in which we have used fresh bronchus and tracheal grafts to close defects. These sections of the trachea have been taken from other dogs. The grafts have been prepared by removing the mucosa and serosal surface and some of them have been prepared in solutions and refrigerated for as long as three weeks. To repair the bronchus we have used a large cylinder of trachea, removing the membranous portion completely. A portion of the main bronchus was resected and this section of trachea placed around both ends of the bronchus so that the continuity of the main bronchus was re-established by a tube of trachea which is completely surrounded by cartilage and, for that reason, fairly rigid. We have observed the dogs for two and one-half months. At present there is evidence that the mucosa is covering the grafts completely, the dogs are well, and we see no evidence of a significant stenosis. However, it will be necessary to keep the dogs eight months to a year, sacrifice them periodically, and at that time we will be able to tell whether the grafts fulfill all the conditions Dr. Gebauer says must be fulfilled. It is our opinion, however, that the grafts merely act as a framework for the mucosa to grow over and for possible regeneration of the other structures of the trachea and bronchial wall.

bronchus and dorsal lobe orifice were normal. The stenosis was most marked at the carina and just proximal to the upper lobe stump. A dermal inlay graft was applied, as depicted in Figs. 9 and 10. It was approximately $\frac{3}{4}$ inch wide and $2\frac{1}{4}$ inches long. The aortic arch was mobilized and retracted to expose the trachea and central portion of the bronchus for

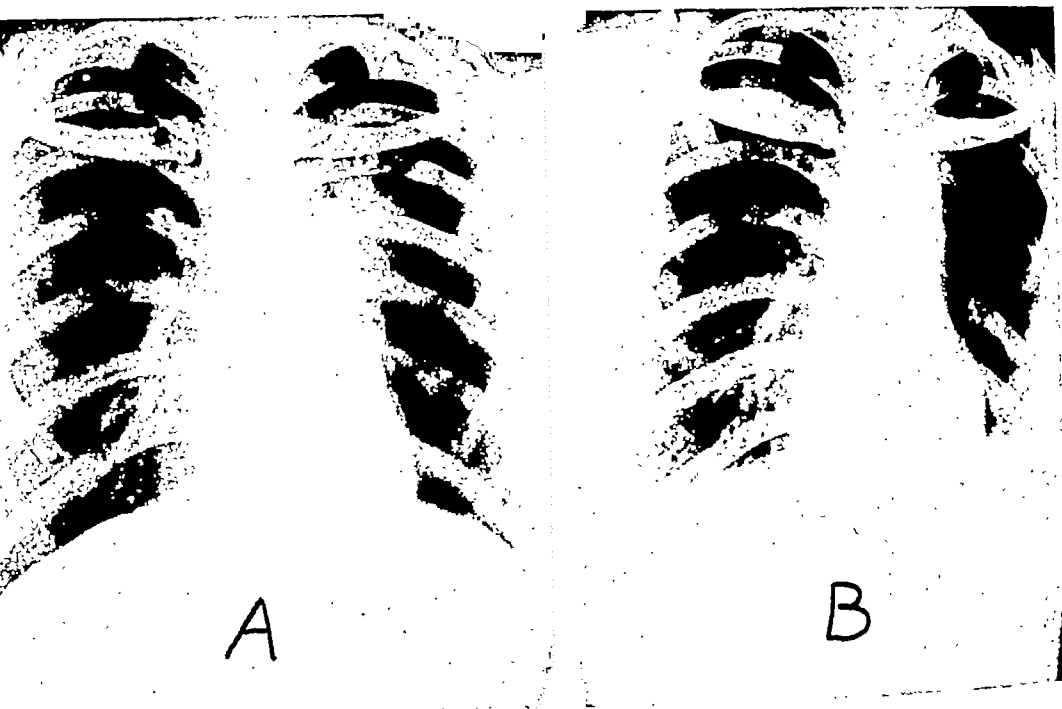


Fig. 17 (Case 4, R. C.).—A, Before operation; small contracted, partly atelectatic, left upper lobe. Sputum negative. Recent increase in the size of the upper lobe density plus increased cough and wheeze occurred. Diminished to absent breath sounds over whole left chest. B, After left upper lobectomy and a dermal graft to the whole main bronchus.

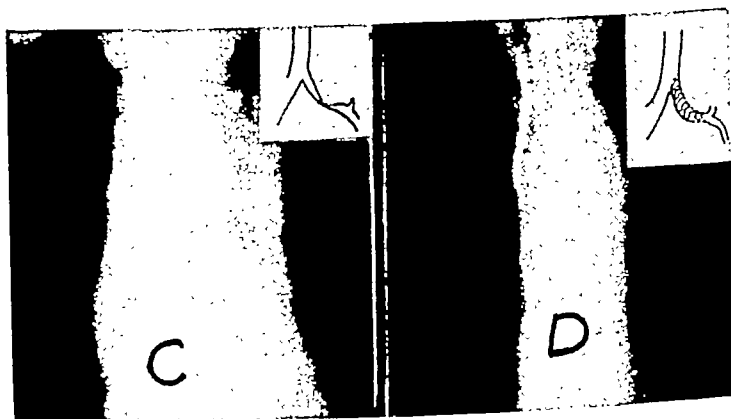


Fig. 18 (Case 4, R. C.).—C, Planigram before operation; stenosis of the left main bronchus, occlusion of the upper lobe bronchus. D, After operation; good bronchial lumen; well-rounded graft. Graft epithelized within five weeks; good, open, round bronchial lumen beyond the carina. Possibly recurring stenosis at the carina.

suture. Retraction of the aorta caused considerable stretch of the recurrent laryngeal nerve. After the graft was applied, the lower lobe inflated and deflated easily.

The highest postoperative temperature was 100.1° F. and pulse 104 on the second day. Both were normal after the fourth day. The lower lobe was completely expanded on the

The following is a report on the status of each of the patients reported in the preceding paper a year or more after operation.

CASE 1.—A. F. (Fig. 20). The upper, extreme end of the wire within the graft was cut and removed from the posterior tracheal wall through a bronchoscope. The bronchus, six months after operation, was clean and glistening. The lumen was oval and small at the distal end of the graft. A 6 mm. dilator was easily passed. The patient was discharged eight months after operation, sputum negative, free of symptoms, and the chest examination was normal. One year after operation the patient is working part time; there are no

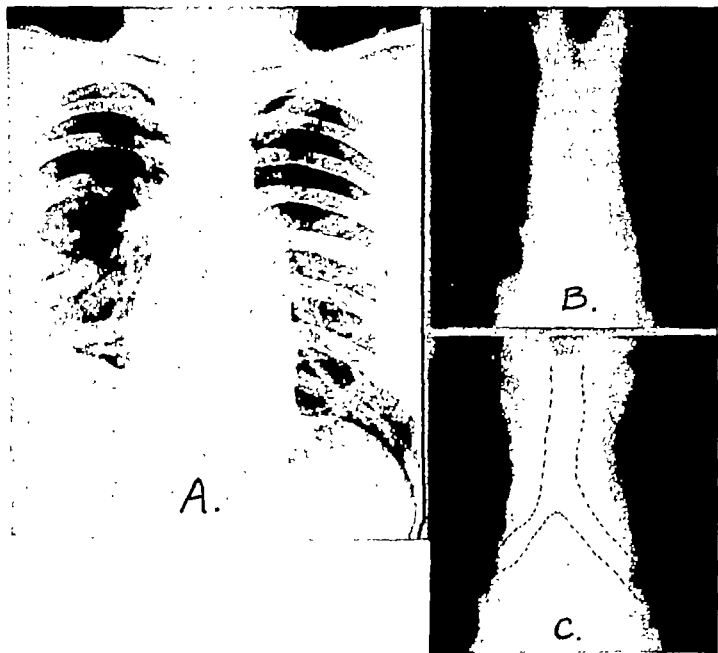


Fig. 20 (Case 1, A. F.).—A, Chest film one year postoperative. B, Unmarked planigram of trachea and bronchi eight months after operation. C, Same, marked.

symptoms. She feels that she can breathe much deeper and easier than before operation. Bronchograms show the right main bronchus to have a caliber about one-half that of the left main bronchus. The grafted bronchus is evidently physiologically adequate, although far from anatomically perfect.

CASE 2.—D. K. (Fig. 21). There were never any significant bronchial symptoms in the postoperative period. Good breath sounds were always audible on the left. A standard 7 by 40 bronchoscope could be passed through a clean, smooth, grafted bronchus and the lower lobe branches inspected. This was difficult because of stiffness of the bronchus and an exaggerated upward curve (Fig. 21, B). The patient professed a marked improvement in breathing capacity. Undoubtedly some of this was due to expansion of the small right pneumothorax. Eight months after operation a modified thoracoplasty was done over the expanded

DR. EDWARD J. O'BRIEN, Detroit.—Thank you for allowing Dr. Jackson to discuss this paper, but this is such an integral part of Dr. Gebauer's talk and it was all at his instigation; none of the ideas were ours, they were all Dr. Gebauer's. I thought it best to have Dr. Jackson talk first.

ADDENDUM AND FOLLOW-UP, NOV. 30, 1949

The reasons for the disparity between the particularly good clinical results, and the sometimes puzzling bronchoscopic findings, have become apparent since the submission of the preceding paper. They are depicted in Fig. 19, and were elaborated in a report already published.*

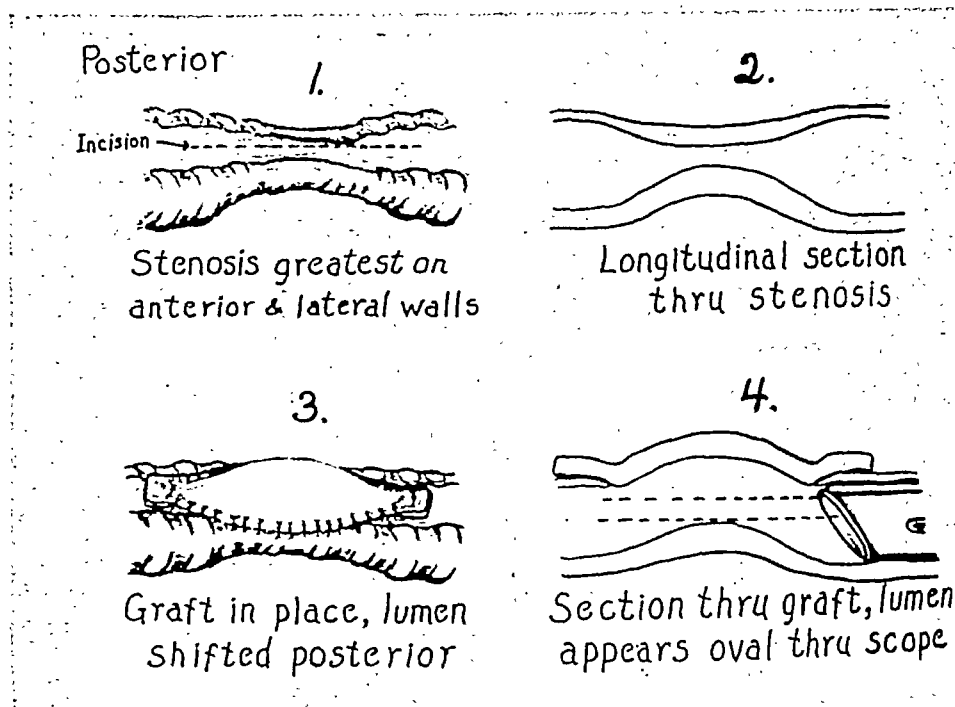


Fig. 19.—Drawing demonstrating the usual transposition of the bronchial lumen by a posterior dermal graft, and the consequent bronchoscopic appearance.

In addition, it seems that a bronchial passage does not have to be normal in caliber and contour to be physiologically adequate. Evidently there is a crucial point in the degree of bronchial stenosis or deformity in each individual case. Until a stenosis reaches this point, lung function is practically normal and there are few significant clinical symptoms. On the other hand, once the crucial degree of stenosis is obtained—whether by fibrous contracture, edema, inflammation, or dried secretions—cough, wheeze, x-ray evidence of expiratory block, or intermittent atelectasis occur, and a gradual process of lung destruction is initiated.

It is quite likely that dermal bronchial grafts will restore stenosed bronchi to normal shapes and dimensions only occasionally. The incidence of such results depends on many factors. Of these, the experience of the operator is highly important.

*Hawaii M. J. 8: 413-418, 1949.

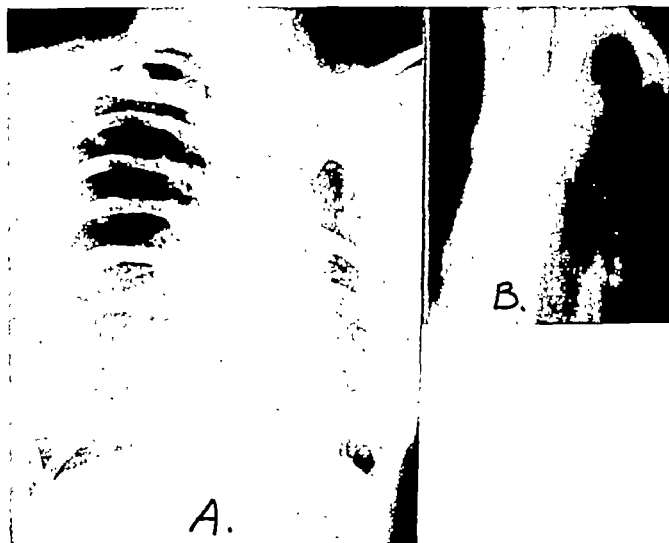


Fig. 22 (Case 3, E. Y.).—A, Chest film one year after bronchial graft, left diaphragm has descended. Note low, flat diaphragm, typical of emphysema. B, Planigram of trachea and bronchi same time.

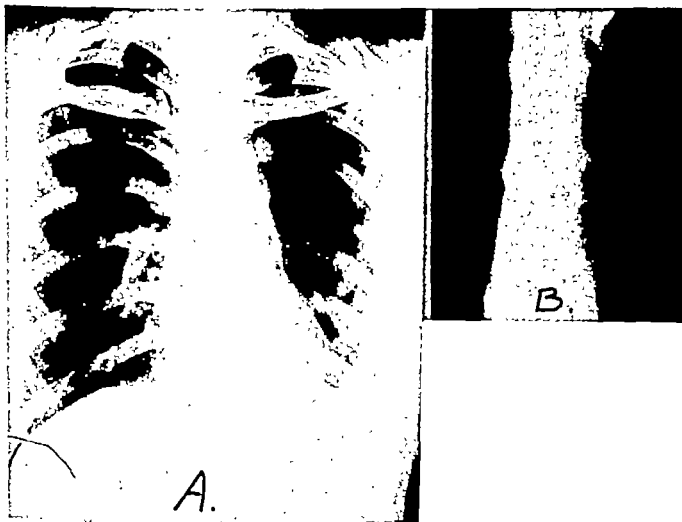


Fig. 23 (Case 4, R. C.).—A, Chest film one year after bronchial graft. B, Planigram of trachea and bronchi same time.

left lower lobe because a light nodular infiltration appeared in its apex and the sputum became positive (Fig. 21, *A*). Cultures were still sensitive to streptomycin and a three-month, 90 Gm. course was administered. The patient had no difficulty with the one-stage thoracoplasty. She was discharged one year after bronchial graft, symptom free, and sputum negative. Physical examination discloses no difference in good bilateral breath sounds. This graft is considered anatomically, as well as physiologically, adequate.

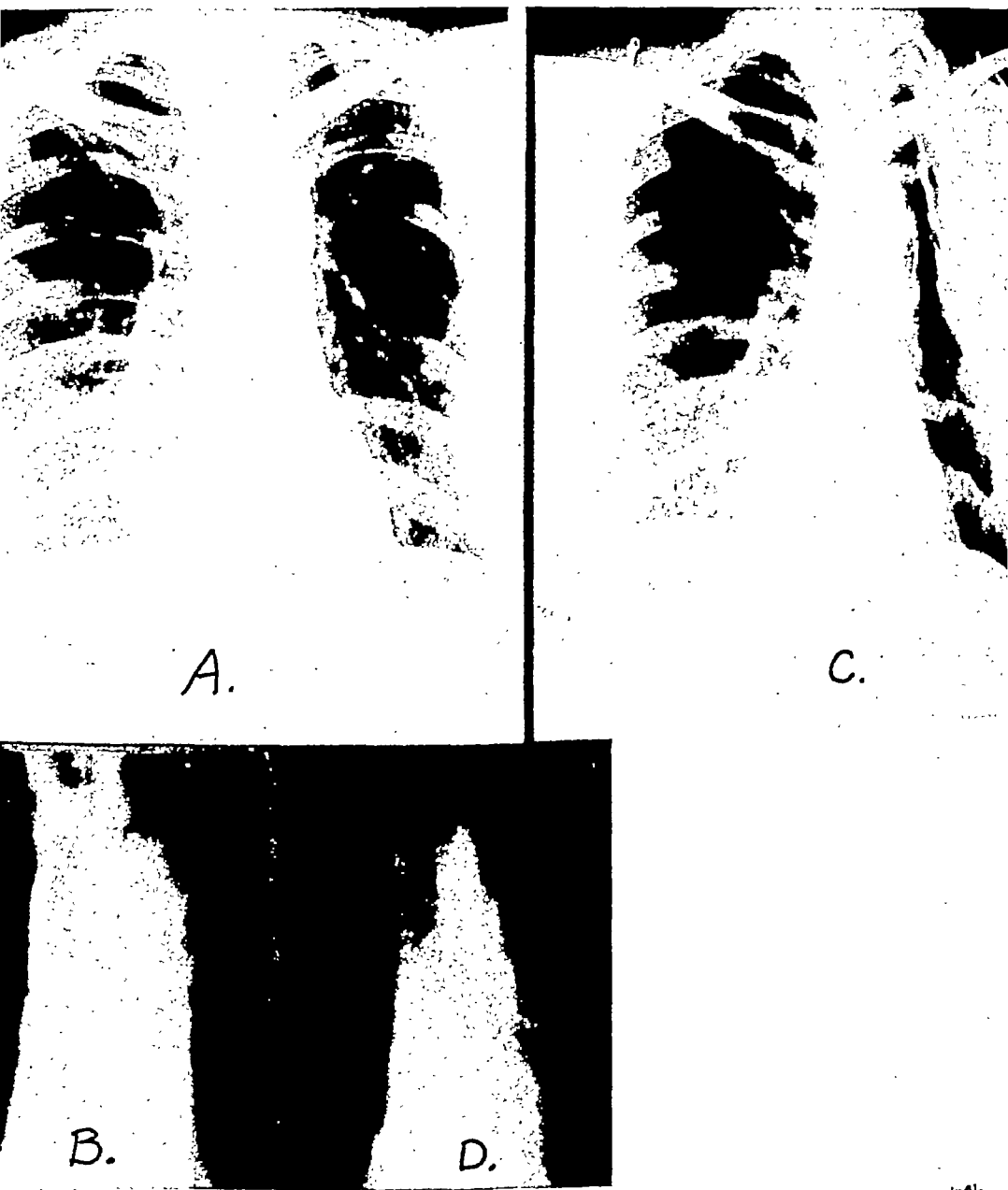


Fig. 21 (Case 2, D. K.).—*A*, Chest film eight months postoperatively, light nodular infiltration expanded apex of left lower lobe. *B*, Planigram of trachea and bronchi at that time. Sputum positive. *C*, Chest film one year after bronchial graft and four months after thoracoplasty. *D*, Planigram same time.

CASE 3.—E. Y. (Fig. 22). Considerable respiratory improvement occurred when the left diaphragm began to function five months after operation. Ventilatory studies indicated a 300 c.c. increase in vital capacity compared to preoperative determinations. They also

EVALUATION OF EXTRAPLEURAL PNEUMONOLYSIS AND PARAFFIN PLOMBAGE FOR CHRONIC PULMONARY DISEASE

H. RYERSON DECKER, M.D.
PITTSBURGH, PA.

THE operation of paraffin plombage following extrapleural pneumonolysis for collapse treatment of pulmonary tuberculosis was proposed by Baer and used first under his direction in 1913, when other materials aimed to stabilize and maintain extrapleural collapse, such as fat, muscles, gauze, had proved unsatisfactory. In the years following it had considerable vogue in Europe. In this country, however, the procedure has never had popularity nor widespread use, judging by the comparatively few reports in the literature. The purpose of this paper is threefold: (1) to review briefly the indications, limitations, and technique of the operation; (2) to report a small series of my own cases, twenty in number; seventeen for pulmonary tuberculosis and three for nontuberculous pulmonary suppuration; (3) to evaluate results as recorded in the literature.

Plombage can be used successfully to accomplish the collapse of a localized tuberculous lesion, and particularly one with a coexistent cavitation. The ideal location is the apex of the lung. The distinct appeal of the operation lies in the fact that it is not ordinarily a hazardous surgical procedure, resulting in mortality, and therefore, may be undertaken in cases of poor surgical risk, either on account of tuberculosis or systemic disease. Furthermore, because of its selectivity, it spares good lung and conserves vital capacity. If plombage fails to accomplish its collapse objective, thoracoplasty may still be utilized.

Plombage can be either an elective primary operation or one supplementary to other collapse procedure. It is chosen in preference to other methods of collapse therapy such as intrapleural pneumonolysis, phrenic crush, or thoracoplasty, when they have been judged either not feasible, not servicable, or too hazardous. In the poor risk group, it may be undertaken to improve the condition of the patient in preparation for a later thoracoplasty. As secondary operation, plombage is used when other methods have been tried and have failed to close cavitation.

Most cases in which plombage is to be used have far-advanced bilateral tuberculosis and demonstrable single or multiple cavities. Experience has shown that the ideal group has a unilateral lesion extending not lower than the second intercostal space, and a cavity not more than 2 cm. in diameter. The scope, however, is much broader and includes the following groups of cases:

1. Unilateral lesion which extends not lower than the second intercostal space
2. Unilateral lesion with low vital capacity

Read (by title) at the Twenty-eighth Annual Meeting of The American Association for Thoracic Surgery, Quebec, Canada, May 31, June 1, 2, 1948.

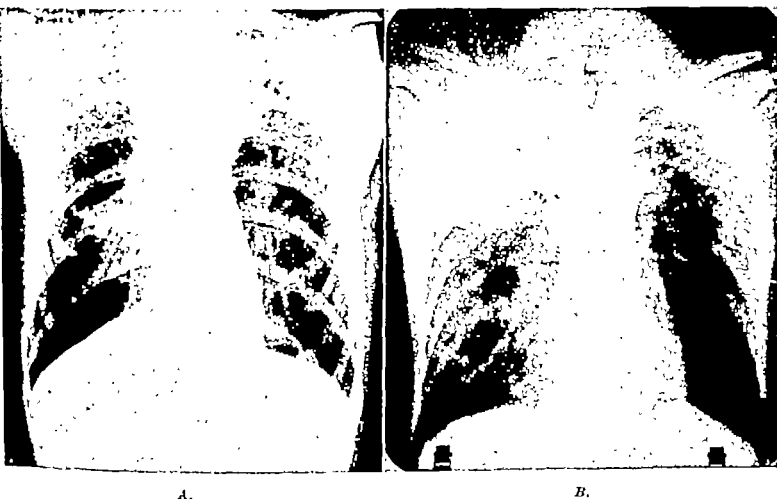
support the clinical impression of severe emphysema, probably present a long time before the first-stage thoracoplasty. She still has dyspnea on exertion. Eight months after operation she was transferred to an ambulant ward preparatory to discharge. A bronchoscope with a 6 mm. lumen could be passed to the branches of the lower lobe without difficulty, but the bronchial wall is rather soft in the region of the graft; otherwise nothing can be observed to distinguish the graft from the remainder of the bronchus. On the ambulant ward the patient had some difficulty with a long walk to meals, and a severe secondary anemia occurred. This was corrected and she is about to be discharged from the hospital. All sputum examinations have been negative since operation.

In summary, it is felt that thoracoplasty was contraindicated in the first place, because of an unsuspected emphysema. Lobectomy has probably arrested or cured her tuberculosis, but has not materially altered her prognosis. The bronchial graft has undoubtedly improved the respiratory function by permitting better use of the left lower lobe.

CASE 4.—R. C. (Fig. 23). Subsequent bronchoscopies revealed a good bronchial lumen over 8 mm. in diameter and apparently lined with clean epithelium. The early pessimism was unwarranted. The protruding wire was cut and removed from the lower trachea. Since then no wire has ever been visualized and an excellent bronchial lumen is present. This patient feels a marked improvement in breathing capacity. As before operation, sputum examinations have remained negative. She was discharged five months after operation. She had no symptoms and normal breath sounds on both sides. The preoperative hoarseness has been increased by the addition of a persistent left vocal cord paralysis to a scarred larynx. One year after operation she is doing all her own housework and is symptom free.

body reaction has formed a connective tissue capsule and adequate septum or shelf beneath the plug and above the collapsed lung to prevent migration of the paraffin. Cavities will diminish in size quickly or gradually to the point of obliteration, depending upon the speed of centrization in the individual case.

My own group of 17 tuberculous patients were largely war veterans in an age group of the fifth and sixth decades, and known to have had tuberculosis for six months to eleven years. All, with one exception, had far-advanced bilateral disease, and all had cavitation. The disease was limited to upper lobes in 13; 4 cases had scattered involvement in other lobes. In 1 case there was a unilateral basilar lesion. The sputum was laden with tubercle bacilli. In only 4 cases was the operation a primary procedure. Eleven had had a previous ineffectual artificial pneumothorax; 2 of these also a phrenic crush. Two had had only a previous phrenic crush. In almost every instance a thoracoplasty was considered, but decided against either on account of the patient's condition as contraindicated at the time or because the patient refused to have it. Two patients had plombage as a last resort, and both did badly. Cavities were unilateral in 8 cases, bilateral in 9, and ranged in size from $1\frac{1}{4}$ to 4 cm. Bilateral operations were done in 2 cases, were proposed, but refused in 3 others. In 1 case the operation was attempted but not completed, because a cleavage line of the pleura could not be obtained.



A.

B.

Fig. 1 (Case 1).—R. D. J., aged 57 years. Refused thoracoplasty; $3\frac{1}{2}$ cm. heavily-walled cavity right apex practically closed by plombage. Sputum converted. A, Before plombage, B, four months later. Bucky exposure.

CASE REPORTS

CASE 1.—R. D. J. Clerk. Age, 57 years. Symptoms for one and one-half years. Admitted Veterans Hospital, Aspinwall, February, 1936. Bilateral upper lobe tuberculosis to fourth spaces. Cavity, right lung, 3.5 cm.; left lung, 2 cm. Sputum positive. Refused thoracoplasty. Plombage, June, 1936. Novocain anesthesia; 350 Gm. pack. Two-inch section third rib. In August, 1936, cavity closed. Sputum negative. Plombage was planned for left apex. Patient went A. W. O. L. Has not been traced.

CASE 2.—P. R. Salesman. Age, 41 years. Symptoms for two years. Admitted Aspinwall, 1935. Bilateral lesion, infiltration both upper lobes with small areas of disease

3. Bilateral lesion with contralateral lung inactive
4. Bilateral lesion with both apices active in good risk cases
5. Bilateral lesion, active tuberculosis in opposite lung

CONTRAINDICATIONS

Plombage will prove disappointing in the following groups:

1. Where the patients are seriously ill with low resistance
2. Where it is undertaken as a matter of last resort
3. Where the disease has extended to the middle or lower lobes
4. Where the disease is acute
5. Where cavities are extremely rigid or more than 4 cm. in diameter.

OPERATIVE TECHNIQUE

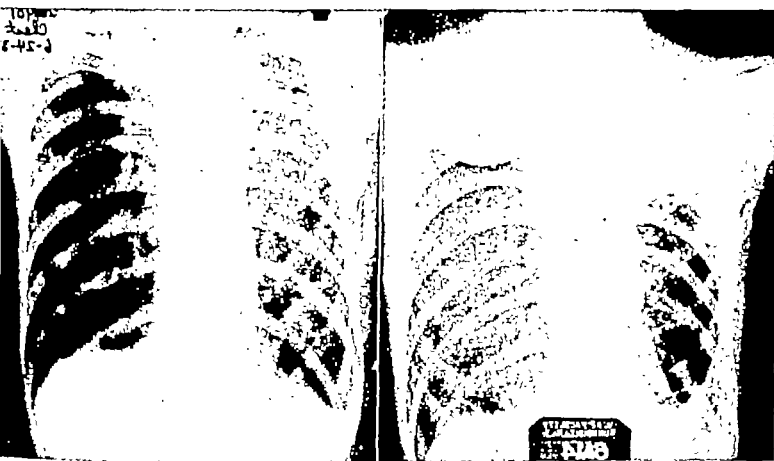
The operative technique is not complex. For apical lesions posterior exposure is preferable to anterior approach, chiefly on account of the better skeletal muscle support. With a paravertebral incision centering just above the spine of the scapula a section of the third or fourth rib when removed will give good access to the apex of the lung. Once the line of cleavage between the parietal pleura and the endothoracic fascia is established, careful gentle dissection with the finger, or a gauze sponge in a holder will mobilize the apex of the upper lobe satisfactorily. Frequently several resistant fascial bands will require sharp division and ligation between clamps. The procedure can be almost bloodless. At times it may be impossible to secure a safe line of cleavage and the operation will have to be abandoned. This happened in one of my cases. At other times difficulty will be found in separating the mediastinal pleura, and it may be the part of wisdom to desist to avoid tearing into the lung or great vessels. Separation should be sufficient to secure good lung relaxation, and apparently to prevent the cavity from being pinned against the vertebral column. Contrariwise separation should not be so extensive that it does not afford a proper supporting bed for the paraffin. Seldom should the separation extend below the level of the fourth rib. There is always a tendency for a cavity to sink downward and inward and escape collapse. Gentle handling is likely to minimize bleeding and subsequent serous exudation, and to prevent perforation of the lung or pleura. Paraffin should act as a passive agent. Pressure effect is to be avoided, unless in the case of scirrhus cavities. Therefore, large packs in excess of 500 Gm. are contraindicated. Usually a pack of 200 to 300 Gm. will be sufficient and will be well tolerated. The actual amount used will depend considerably on the size of the cavity, the resistance of the lung, and the character and extent of the disease. After paraffin with a melting point of 52° F. or more is placed in the prepared cavity, muscle planes should be accurately closed to help prevent any extrusion of the pack. Careful asepsis pays dividends, but does not prevent infection of the field from a pulmonary or pleural focus. Added safety comes now in the use of antibiotics, as when penicillin and streptomycin are instilled into the wound, and administered intramuscularly before and after operation. Should infection develop, early removal of the pack is indicated with appropriate treatment such as Dakinization of the infected cavity or injection of the antibiotics locally.

The choice of anesthesia depends upon the individual case: taking into consideration the general physical condition, the gravity of the pulmonary lesion, and mental reaction. My own practice has been to use local Novocain anesthesia and to supplement this as indicated with small amounts of intravenous pentothal or with a basic dose of Avertin. In recent years, however, I have practically discarded the use of Avertin for all surgery. It is my conviction that at the close of any operation for pulmonary disease, or soon thereafter, it is important for the patient to cough and take deep breaths. Local anesthesia allows this.

Postoperative course is usually uneventful. Fever of several degrees for several days is to be expected. Rest in bed is advisable for a period of four or five weeks, until a foreign

thoracoplasty. Plombage, December, 1937. Novocain anesthesia. Two-inch section third rib right; 500 Gm. pack. March, 1938, cavity closed apex. Refused operation on left side. Discharged July, 1938. Sputum plus zero. Readmitted July, 1939. Cavity $3\frac{1}{2}$ cm. upper lobe below paraffin pack. Right upper lobe 3 cm. cavity. Sputum positive. Transferred to Hopemont. Died December, 1940, of progressive tuberculosis.

CASE 8.—R. W. H. Age, 40 years. Laborer. Symptoms for two years. Admitted to Veterans Hospital, Aspinwall, September, 1937. Tuberculosis bilateral involving upper two-fifths of left lung and upper one-half of the right lung. Bilateral cavitation 3.5 cm. Artificial pneumothorax instituted on right side. Secured only 25 per cent collapse. Plombage, right, January, 1938. Novocain anesthesia. Two-inch section third rib; 500 Gm. pack. Sputum converted to plus zero. Following operation developed recurrent hemoptysis. April, 1938, phrenic crush. Did not stop hemoptysis. Roentgenogram showed no diminution of size of right cavity. Progressive extension of disease in both lungs. Death May, 1938.



A.

B.

Fig. 2 (Case 6).—M. H. C., aged 44 years. Bilateral plombage. Marked clinical improvement. Sputum converted. A, Before plombage. B, Eight months after left and four months after right plombage.

CASE 9.—S. M. Age, 39 years. Negro porter. Tuberculosis since 1934. Admitted Aspinwall, September, 1937. Bilateral pulmonary tuberculosis involving both upper lobes as far as the second interspace. On left side a $3\frac{1}{2}$ cm. cavity, on right 1 cm. Sputum positive. General condition good. Artificial pneumothorax instituted on left side. Partial collapse, but cavity not affected. Plombage, May, 1938. Novocain Avertin anesthesia. Section $1\frac{1}{2}$ inches right fourth rib. Roentgenogram September, showed cavity 1 cm. in diameter. February, 1939, no cavity seen. Sputum negative. Forty pounds gain in weight. Artificial pneumothorax continued on left side. Discharged May, 1939, with maximum hospital benefit to return to outpatient department for continuation of artificial pneumothorax. Readmitted June, 1940. Spitting up paraffin. Both upper lobes showed marked fibrosis, especially about the right apical pack and a small fibrotic area in right apex, indicating closed cavity. Sputum negative. August, 1941, developed a spontaneous collapse of right lung from which he made a good recovery, but a spread of the disease in the right upper lobe was noted. Later progressive extension in both lungs. Apparently a new cavity developed in the base of the left upper lobe. Death in April, 1942.

in the lower lobes. 4 cm. cavity, left upper lobe. Sputum positive. Artificial pneumothorax begun left side, 1935; phrenic crush, February, 1936. Some improvement in the lung. No effect on cavity. Plombage, September, 1936. Novocain Avertin anesthesia. Two-inch section third rib. 350 Gm. pack. November, 1936, roentgenogram showed cavity closed. Sputum negative. January, 1938, transferred to Sunmount. Discharged April, 1942, maximum benefit. No active tuberculosis. Cavity closed by plombage. Sputum negative on guinea pig inoculation. Died in 1943, whether from tuberculosis is not known.

CASE 3.—J. T. C. Age, 52 years. Restaurant operator. Symptoms two years. Admitted Veterans Hospital, Aspinwall, 1935. Bilateral upper lobe tuberculosis with scattered areas in all lobes. 4 cm. cavity, left upper lobe. General condition poor. Sputum positive. Artificial pneumothorax right side; secured 50 per cent collapse; left side, 25 per cent collapse. No effect on cavities. Plombage March, 1937. Novocain anesthesia. Two-inch section third rib removed; 300 Gm. pack. Roentgenogram showed April, 1937, cavity apparently collapsed. Sputum remained positive. Died May, 1938, of progressive tuberculosis.

CASE 4.—E. T. Negro. Mill worker. Age, 42 years. Symptoms since January, 1935. Admitted to Aspinwall October, 1935. Spotty infiltration all lobes bilaterally; 7 cm. cavity in left upper lobe and 2¼ cm. cavity in right upper lobe. Sputum positive. Artificial pneumothorax instituted on right side. Discontinued in five months. Plombage, right, September, 1937. Novocain anesthesia. Small sections third and fourth ribs; 250 Gm. pack. Refused operation for left-sided cavity. Roentgenogram September, 1938, showed cavity in right lung closed. Sputum still positive. Generalized extension of tuberculosis. Died December, 1938, with left-sided pyopneumothorax.

CASE 5.—M. H. Repairman. Age, 42 years. Onset of tuberculosis, 1930. Treated Castle Point 1932 to 1936. Admitted to Aspinwall December, 1936. General condition good. Bilateral tuberculosis, infiltration of right upper lobe to fifth space with 3 cm. cavity; left upper lobe to fourth space. Sputum positive. Artificial pneumothorax begun on right side. Discontinued December, 1937. Plombage right apex August, 1937. Novocain anesthesia. Two-inch resection of third rib; 500 Gm. pack. General clinical improvement. Twenty-pound gain in weight. Sputum converted to plus zero. October, 1937, roentgenogram showed cavity closed, no evidence of active disease in either lung, pleural thickening, right costophrenic sinus obliterated; upper one-third left lung, well-organized fibroid deposit. Admitted Sunmount, March, 1939. No active disease in either lung. Sputum negative. Has not been traced since discharge with maximal hospital benefit.

CASE 6.—M. H. C. Machinist. Age, 37 years. Onset of tuberculosis, 1931. Admitted Aspinwall, August, 1931, and subsequently three times. August, 1937, bilateral tuberculosis involving upper two-thirds of left lung, and apex of right lung where there was a 3 cm. cavity; a smaller cavity 1½ cm. in left apex. Sputum positive. Artificial pneumothorax instituted on left side proved inefficient. General condition poor, emaciated, and toxic, and not suited for thoracoplasty. Plombage January, 1938. Novocain and Avertin anesthesia. Two-inch section of fourth rib; 500 Gm. pack. Roentgenogram April, 1938. Cavity right lung closed. Some improvement in general condition. May, 1938, plombage for left apical cavity. Novocain and Avertin anesthesia. Two inches fourth rib; 500 Gm. pack. Condition remained static. Transferred to Sunmount, August, 1938. Roentgenogram showed no cavitation in either apex. Adhesive pleuritis small areas of well-organized fibrosis in left lung. Sputum negative. September, 1938, began to spit up small bits of paraffin. November, 1938, paraffin pack removed by Dr. Welles. Was discharged from Sunmount as having maximal hospital benefit, 1940. Untraced since then.

CASE 7.—J. H. M. Salesman. Age, 44 years. Onset of tuberculosis, 1927. Artificial pneumothorax had been tried and failed in hospital, Hopemont, W. Va. Admitted to Aspinwall, January, 1937. Bilateral pulmonary tuberculosis, upper one-third left upper lobe with 2 cm. cavity, upper one-fourth right upper lobe 1½ cm. cavity. Sputum positive. Refused

positive. Plombage, February, 1934. Sodium pentothal Novocain anesthesia. Two-inch section of fourth rib; 400 Gm. pack. Developed *Staphylococcus aureus* wound infection, bronchopneumonia bilateral, and bacteriemia. Paraffin pack removed. Wound Dakinized. Death six days after operation. Autopsy showed no evidence of bronchopleural fistula. Cavity collapsed partially.

CASE 13.—M. S. In 1920 was struck in the right chest. Had hemoptyses for six months at irregular intervals and pain in right chest on exertion. Dec. 27, 1929, developed severe hemoptysis controlled by right phrenic crush. Roentgenogram showed some fibrosis and small areas of infiltration in the right upper lobe. In the next two years still subject to small hemoptyses, and pain in the chest. Dec. 21, 1933, developed very severe bleeding over three days' time. Plombage, Jan. 1, 1934. Novocain anesthesia. Three-inch section of eighth rib in the posterior axillary line; 500 Gm. pack. This controlled the bleeding. Admitted to Tuberculosis League Hospital January, 1935, on account of persistent fatigue, night sweats, and dyspnea on exertion. Roentgenogram showed moderately dense mottling



Fig. 4 (Case 13).—M. S. Paraffin plombage for lower lobe lesion fourteen years previously. Apparently cured.

in the second and third spaces, paraffin pack below the level of the third rib. Only one sputum examination in March, 1935, returned positive for tuberculosis. Subsequent examination showed increased density in the right lung field. Repeated sputum examination proved negative. Hospital care continued at Cresson Sanatorium for one year. Discharged September, 1937. Returned to his native Czechoslovakia to work on farm. Pressed into service in German Labor Battalion for four years. Returned to this country January, 1948, to take up farming. Free from symptoms. Looked to be in perfect physical condition. Roentgenogram showed no evidence of active tuberculosis. Left lung field entirely clear. Dense fibrosis from the third rib to the base of the right lung.

CASE 10.—H. O. H. Age, 48 years. Clerk. Onset of tuberculosis, 1934. Admitted Aspinwall, April, 1938. Bilateral tuberculosis, infiltration of right upper lobe with 1½ cm. cavity; fibrosis of left apex. Sputum plus zero. Clinically an associated bronchitis. Refused thoracoplasty. Plombage, October, 1939. Novocain anesthesia. Two-inch section third rib; 150 Gm. pack. Pleura was accidentally opened, followed by a marked collapse of the lung and dyspnea. Relieved by aspiration of 1,000 c.c. air. April, 1940, roentgenogram showed cavity to be closed. Sputum negative. Readmitted January, 1943, on account of asthma and dyspnea. Roentgenogram showed no evidence of active tuberculosis. Sputum negative. Readmitted October, 1947, on account of asthma. No evidence of an active tuberculosis. Sputum negative. April, 1948, only symptoms due to asthma and chronic bronchitis.

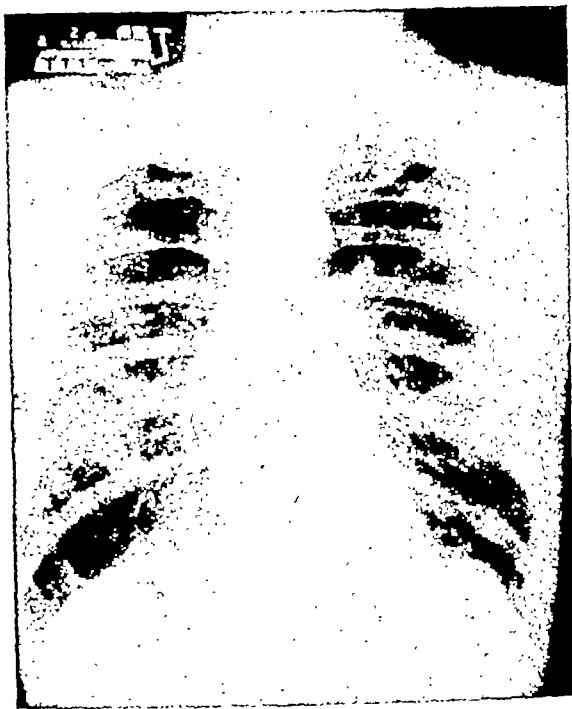


Fig. 3 (Case 10).—H. O. H., aged 48 years. Plombage, April, 1939, closed 1.5 cm. cavity right apex. Clinically arrested. Sputum conversion. February, 1947, no active tuberculosis. Clinically arrested.

CASE 11.—J. C. Age, 44 years. Negro. Steelworker. Symptoms for eight months prior to admission November, 1936, Aspinwall. Hemoptysis frequent. Sputum positive. Wassermann + 4. Bilateral infiltration of both upper lobes as far as second interspace, 2 cm. cavity on right side. Artificial pneumothorax induced on right. Poor collapse. December, 1936, phrenic crush, did not secure a rise in the diaphragm. Plombage May, 1937. Novocain Avertin anesthesia. Two-inch section third rib; 500 Gm. pack. Roentgenogram June, 1937, showed collapse of cavity. Sputum negative. No further hemoptysis. Discharge improved. Readmitted October, 1940, because of hemoptysis. Roentgenogram showed right upper lobe cavity closed, extensive fibrosis in both lungs, a spread of tuberculosis in the left upper lobe. Death May, 1941, from progressive tuberculosis.

CASE 12.—L. R. Salesman. Age, 34 years. Symptoms of tuberculosis, chiefly pleuritic pain, for two years. Admitted to Tuberculosis League Hospital. Roentgenogram showed scattered areas infiltration at right apex and in the third and fourth spaces; on left side soft infiltration in the second, third, and fourth spaces, and a 5 cm. apical cavity. Had carried artificial pneumothorax for over a year with only 30 per cent collapse. Sputum

Married. Delivered of normal child by cesarean section, 1942. For seven years has taken care of five-room house and enjoyed good health with no pulmonary symptoms. Roentgenogram April, 1948, shows no evidence of active tuberculosis. Cavity closed. Paraffin pack still in place.

CASE 17.—L. L. Age, 22 years. History of tuberculosis two years. Frequent hemoptysis. Roentgenogram showed bilateral tuberculosis both upper lobes. Cavity in right upper lobe 6 cm; left upper, 5 cm; general condition not good. Plombage, left, May, 1943. Cyclopropane Novocain anesthesia undertaken as a forlorn hope. Two inches of third rib removed; 350 Gm. pack. Roentgenogram showed partial closure of the cavity. Plombage, right, July 10, 1943. Novocain and cyclopropane anesthesia 500 Gm. pack. Two inches of fourth rib removed. Following these operations there was no further hemoptysis. Sputum continued profuse and positive. Progressive involvement of both lungs. Death in October, 1944. Neither cavity was completely obliterated. February, 1944, patient commenced to expectorate paraffin in small amounts. Shortly thereafter a bronchopleural fistula developed on the right side, and through this at times small bits of paraffin were extruded. Eventually this fistula closed.



A.

B.

Fig. 6 (Case 20).—C. B., aged 28 years. A, Abscess lung. Before pack, 1935. B, Taken March, 1948. No pulmonary symptoms after pack.

CASE 18.—W. W. Miner. Age, 43 years. Admitted Aspinwall, August, 1938, with symptoms of pulmonary suppuration. Bronchoscopy showed no evidence of tumor. In November developed an empyema containing *Staph. aureus* and *Streptococcus hemolyticus*. When empyema had partially cleared several abscesses in the lung were drained. In April, 1939, developed severe hemoptysis which was not controlled by a phrenic crush. May 2, 1939, plombage of right lower lobe. Novocain anesthesia. One and one-half inches of eighth rib removed posterior axillary line. Hemoptysis was not controlled. Death occurred May 13, 1939, from large hemorrhage. Autopsy disclosed bronchiogenic carcinoma of lower lobe bronchus.

CASE 14.—M. I. C. Age, 44 years. Four-year history bilateral apical tuberculosis. Infiltration in both apices with 2 cm. cavity right apex. Sputum positive. Artificial pneumothorax induced. At the end of four months collapse was poor. Plombage was attempted July, 1937. Avertin Novocain anesthesia. Section two inch, third rib. Line of pleural cleavage could not be obtained. Died of progressive tuberculosis in February, 1938.

CASE 15.—M. S. Female, age, 30 years. Telephone operator. Tuberculosis for eleven years, in which time artificial pneumothorax had been ineffectual, and then had frequent recurrent hemoptysis. Involvement was massive in right apex through the first and second spaces with multiple small cavities. Sputum positive. Plombage January, 1939. Anesthesia sodium pentothal and Novocain. Section one and one-half inches of fourth rib; 375 Gm. pack, securing complete collapse of upper lobe. Patient's condition seemed satisfactory as she left operating room, though cyanosis was noted. Several hours later developed extreme dyspnea, cyanosis, very rapid heart action, and died six hours after operation. Autopsy not granted. It is presumed that the cardiorespiratory failure was induced by too heavy pressure of the pack on the great vessels.

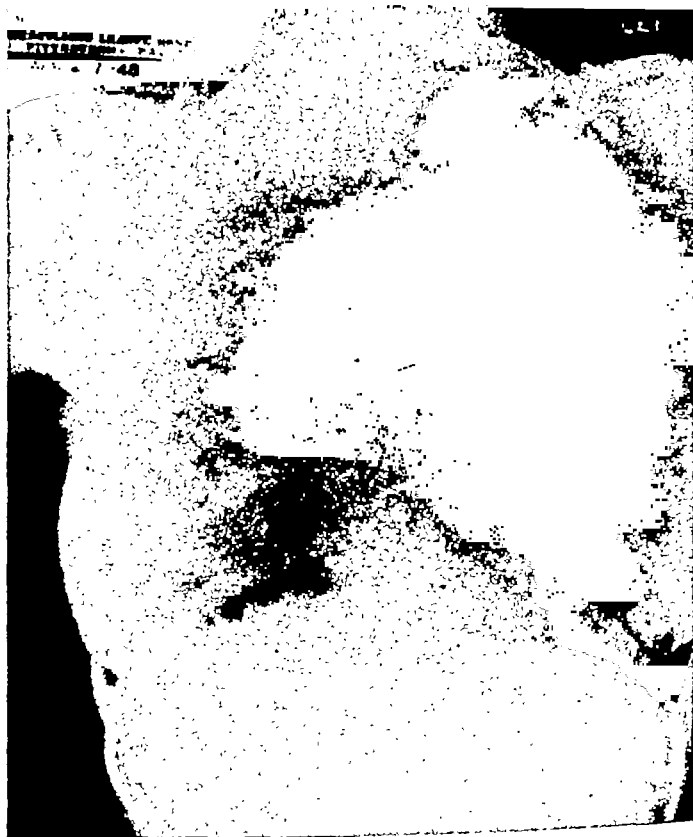


Fig. 5 (Case 16).—E. O. B., aged 25 years. Paraffin plombage fifteen years previously. No pulmonary symptoms. Apparently arrested.

CASE 16.—E. O. B. Female, age, 25 years. Clerk. History of tuberculosis for four years. Bilateral apical infiltration. Cavitation right apex. Positive sputum. Artificial pneumothorax induced in April, 1933. Inefficient. Plombage, November, 1933. Sodium pentothal Novocain anesthesia. Two-inch section fourth rib; 500 Gm. pack. Phrenic crush November, 1935. February, 1936, 2 cm. cavity right lung closed. Tuberculosis left apex arrested. Sputum negative. This patient has remained well. Had an appendectomy, 1938.

TABLE I. POSTOPERATIVE COMPLICATIONS

	MC INDIE, STEELE, AND ALEX- ANDER	HEAD	OTTAVI- ANO	STEELE	DECKER	TOTAL
Superficial wound in- fection	3					3
Deep wound infection		1	4		1	6
Septicemia (fatal)					1	1
Extra pleural effusion	2			1		3
Hemorrhage, extra pleural space	2					2
Cold abscess	3					3
Fistulas, broncho- pleural	5	3	5	2	4	19
Fistulas, broncho- cutaneous			5			5
Fatal lung tear	2					2
Contralateral spread	3	1				4
Paraffin coughed up	1	3	4		4	12
Paraffin removed		1	0		2	3
Transient dyspnea	12					12
Cardio respiratory fail- ure (postoperative)					1	1

CAUSES OF DEATH: POSTOPERATIVE

	MC INDIE, STEELE, AND ALEXANDER	HEAD	BEHRENS	DECKER
Septicemia			3	1
Wound infection		1		
Nontuberculous pneumonia		1		
Cardiorespiratory	1			1
Lung tear	2			
Cold abscess	2			
Operation	2			
Tuberculous enteritis		1		

Staph. septicemia. In a small series incompletely reported by Behrens there were 3 deaths from septicemia.

2. A tuberculous abscess developed about the pack in 3 cases.

3. Fistulas developed, bronchopleural in 19 and bronchocutaneous in five. Eleven patients coughed up paraffin after a period of several weeks to months. The handling of these cases presents somewhat of an individual problem. It may be necessary to remove the pack. Others will stop coughing up paraffin spontaneously. I believe the majority will tolerate the pack for years without unfavorable reaction. Whether this will be true of the newer pack materials, Lucite and fiberwool, remains to be demonstrated.

4. Effusions of size requiring tapping occurred in 3, and a sizable hemorrhage in the cavity in 2 patients.

5. Fatal lung tear happened in 2 cases.

6. Cardiorespiratory symptoms were reported as severe dyspnea in twelve cases by John Alexander. Circulatory failure was responsible for death in two cases.

7. Spread of tuberculosis in the contralateral, or same lung, are hard to evaluate in terms of being due to the operation, since they may represent a

CASE 19.—H. S. Age, 41 years. Salesman. 1930, developed a right lobar pneumonia. Following this had persistent cough and purulent sputum in small amount with occasional small hemorrhage due to bronchiectasis. Admitted to Presbyterian Hospital Feb. 26, 1942, on account of severe almost exsanguinating hemorrhage. Phrenic crush and artificial pneumothorax carried out simultaneously. This failed to control bleeding. Plombage, February 28. Novocain anesthesia. Two inches of eighth rib excised in posterior axillary line; 450 Gm. pack, re-enforced with iodoform gauze pack. No further bleeding. Ten days later iodoform gauze removed and one pint turbid sterile fluid evacuated together with several loose pieces of paraffin. Discharged from hospital April 5 with wound healed, free from cough and sputum. Remained well until July, 1947, when he had small hemorrhage and in September a larger one. Then had a long deferred lobectomy by Dr. Kipp at Mercy Hospital and is free from symptoms.

CASE 20.—C. B. Female, housewife. Age, 28 years. Following dental extraction March, 1934, pneumonia and lung abscess developed. Persistent cough and purulent sputum. Hemoptysis began six months later and recurred at frequent intervals. X-ray examination May, 1935, disclosed a 2 cm. cavity near the periphery in the right lower lobe. Plombage, June, 1935. Novocain anesthesia. Two-inch section sixth rib posterior axillary line; 300 Gm. pack. No further hemoptysis. Cavity closed and patient free from symptoms in several months. Has remained well. Roentgenogram March, 1948, showed pack in place. Lung otherwise normal.

RESULTS

At the present time, of the 16 patients in which plombage was completed, 3 are known to be living and well and free from symptoms after a period of eight, thirteen, and fifteen years. One has married and borne a child; another served in the German Army for four years in the last war. Ten patients are known to be dead. Two died in the immediate postoperative period, one of cardiorespiratory failure and the other of *Staph. septicemia*. The other 8 died as a result of progressive tuberculosis. At least 4 of these after the operation showed immediate and substantial clinical improvement with reduction of cough, sputum, gain in weight, and even sputum conversion, only to relapse in a one- to three-year period. Three of the patients have not been traced beyond a three-year period after operation.

Cavities were completely closed as far as one could judge by x-ray findings in 10 cases, were partially closed in four, and presumably not affected in the 2 patients who died soon after operation.

Four cases were known to have developed a bronchopleural fistula because of paraffin spitting. Hemoptysis in 2 of the patients, persistent before operation, was stopped, in 2 others was not controlled. One case developed recurring hemoptysis after operation. Sputum conversion to negative occurred in 8, to plus minus in 3, and was not obtained in five. In 2 cases of conversion, sputum became positive after one year.

As greatly as one would like to believe that plombage is free from complication and without immediate mortality, it is universal experience that there is morbidity and an occasional death directly due to the operation. I have tabulated the complications as reported in four series of cases totaling 186 patients, including my own 16, in Table I:

1. It will be seen that pyogenic infection is the most frequent complication, superficial in 3 instances and deep in six. One of these cases developed a fatal

normal progress of the disease. In 3 cases at least the spread was thought to be definitely a consequence of the operation.

Reports in the literature of the end results to the patient after paraffin pack are rather meager and incomplete, and cover as a rule only short follow-up periods seldom beyond a year or two. Five reports are tabulated in Table II. Also, the effect of plombage upon cavity closure is infrequently recorded. McIndoe, Steele, and Alexander, however, analyzed the fate of cavities in their 82 cases. They concluded that the closure of cavities less than 2 cm. in diameter will occur in more than 50 per cent of cases, and of cavities more than 2 cm. in diameter in less than 50 per cent; if above 4 cm. in diameter, closure of only 33 per cent may be expected. Supraclavicular cavities have a two-to-one chance and infraclavicular a two-to-three chance of closure, very fibrous cavities less than 50 per cent, honeycombed only 50 per cent. In my own series only 2 cavities out of 14 were larger than 4 cm. and neither of these was closed; 5 out of 6 where the diameter was 1 to 2 cm. were closed; and 5 out of 7 where the diameter was 2 to 4 cm. were closed. In 2 cases only was the cavity supraclavicular, and these were both closed.

Of the 264 cases reported in the table, 31 per cent were regarded as apparently cured, 27.5 per cent in a state of arrest, 12 per cent improved, while 17.5 per cent were unimproved or dead. About 60 per cent then are credited with having benefited markedly from the plombage. Even though with the passage of time, the percentage of good results lessens appreciably, the statistics seem to demonstrate the real value of the operation and justify the confidence of the surgeons who have practiced it.

I have had occasion to place a paraffin pack in 3 nontuberculous patients where hemoptysis was alarming and might easily have been fatal. The first patient had a small abscess, bleeding persistently, located in the upper outer portion of the lower lobe. The resection of the lobe seemed to be too major a procedure and drainage would have required two stages and injured much pulmonary tissue. The second patient had severe bleeding from a lower lobe bronchiectasis, which was not controlled by a phrenic crush and artificial pneumothorax. A segmental resection was not justified on account of the patient's condition. The third patient was known to have pulmonary suppuration, but found at autopsy to have carcinoma of the lung as a primary lesion. In only this case was bleeding not controlled.

I am not one to advocate plombage as a means of treating pulmonary suppuration, when segmental resection or lobectomy can be performed. Occasionally, however, plombage can be of service in healing a small abscess or in tiding over a critical situation until a more favorable time comes for extirpation of the diseased lung.

CONCLUSION

In evaluation of the operation of paraffin plombage in the light of comparatively small experience reported, I believe that this treatment should have a definite place in collapse therapy, and that it is entitled to wider use. It should not be in competition with any other method, particularly thoracoplasty.

TABLE II

	MC INDIE, STEELE, AND ALEXANDER	HEAD	OTTAVIANO	STEELE	BEHRENS	DECKER	TOTAL
	1939	1937	1941	1946	1933	1948	
Year of report	2 to 5 yr.	Several				8 to 15 yr.	
Time of follow-up	31 (37.8%)			27 (69%)	1 to 3 yr.	3 (18.7%)	83 (31%)
Apparently cured	23 (28.1%)		27 (60%)		22 (57%)		50 (19%)
Apparently arrested	9 (11%)		12 (26.7%)	2 (5%)			23 (8.7%)
Quiescent	4 (4.8%)	1 (47.7%)			7 (19%)		12 (32%)
Improved	2 (2.4%)	4 (9%)			4 (10.8%)		10 (3.8%)
Unimproved	7 (8.6%)	3 (6.8%)	6 (13.3%)	5 (13%)	5 (13.2%)	10 (62.5%)	36 (13.6%)
Dead		16 Sputum negative		5 (13%)			21 (8.9%)
Unevaluated							
Untraced	6 (7.3%)					3 (18.7%)	9 (3.5%)
Total	82	44	45	39	38	16	264
Sputum conversion	67%	36.4%	75%	75%		50%	61%
Cavity closure	68.9%					62.5%	65.2%

CHONDROSARCOMA OF THE POSTERIOR MEDIASTINUM WITH HOURGLASS INVOLVEMENT OF THE SPINAL CANAL: RESECTION AND RECOVERY

REPORT OF A CASE

WILSON WEISEL, M.D., AND WILLARD B. ROSS, M.D.
MILWAUKEE, WIS.

THE unusual occurrence of a posterior mediastinal chondrosarcoma with hourglass involvement of the spinal canal as well as invasion of the lung, ribs, and thoracic vertebrae has been the chief reason for reporting the following case.

CASE REPORT (No. 107001).—A. L. was a 40-year-old white veteran, a farmer, admitted to this hospital on Aug. 20, 1948, because of numbness of the legs and inability to walk for the past three weeks. Eight months prior to admission he had noted for the first time a recurring intermittent "knife-like" pain in the left side of the back "at the level of the heart." About one month prior to entry he had noted occasional numbness of the soles of the feet; three weeks before admission this symptom had suddenly increased to numbness of the lower portion of the body below the waist, associated with inability to walk because of weakness of the legs. He had lost about fifteen pounds during the three months preceding his admission. On the day of admission he was unable to void for the first time.

A survey of his past history revealed that in 1944, while serving with the Army in Burma, a tree fell across his back causing him to be hospitalized for sixty-five days. There were no fractures or neurologic disturbances occasioned by this injury however, and the exact nature and extent of the injury was not known. The system review was otherwise noncontributory.

Physical examination revealed a well-developed, well-nourished man lying flat in bed in no apparent distress. The temperature was 98.4° F., the pulse rate 88 per minute, the respirations 20 per minute, and the blood pressure was 134 mm. systolic and 84 mm. diastolic in both arms. The head and neck were normal. The eye examination revealed pupils equal in size and regular in outline; the light and accommodation reflexes were normal as were the extraocular movements; the fundi were normal.

Examination of the chest showed a slight bulge with no distinct outline in the left mid-thoracic paravertebral area which was moderately tender to deep pressure. The heart and lungs presented no abnormal findings.

Neurologic evaluation revealed a marked weakness of both lower limbs. There was a loss of cutaneous sensation below the level of the seventh dorsal segment on the right and below the sixth dorsal segment on the left. The knee and ankle jerks were exaggerated bilaterally, and the Babinski reflexes were positive bilaterally. There was absence of the position sense in the toes of both feet. A study of the spinal fluid by means of lumbar puncture revealed a clear fluid with a complete subarachnoid block. The initial pressure was 190 mm. of water.

The significant findings on roentgenologic examination were the presence of a round circumscribed mass in the posterior left chest and erosion of the left pedicles of the adjacent sixth and seventh thoracic vertebrae (Figs. 1 and 2). Myelographic studies with Pantopaque revealed a spinal cord compression at the level of the sixth and seventh vertebrae.

From the Department of Surgery, Veterans Administration Hospital, Wood, Wis., and The Marquette University School of Medicine.

Published with permission of the Medical Director, Department of Medicine and Surgery, Veterans Administration, who assumes no responsibility for the opinions expressed or conclusions drawn by the authors.

Received for publication April 7, 1949.

When one follows the indications closely, cavity closure and sputum conversion may be expected in one-half of the cases. Many cases will go on to arrest and recovery directly as the result of the plombage. Others are put in condition to survive a secondary thoracoplasty and then go on to recovery. A sizable group will register general clinical improvement for a prolonged period, even though they ultimately succumb to the disease.

REFERENCES

1. Baer, G.: München. med. Wehnschr. 60: 1587, 1913.
2. Baer, G.: Ztschr. f. Tuberk. 64: 319, 1932.
3. Behrens, W.: Tubercule 15: 97, 1933.
4. Bérard, L., and Henry, M.: Lyon chir. 33: 738, 1936.
5. Cutler, J. W.: Am. Rev. Tuberc. 38: 62, 1938.
6. Fruchaud, H.: Rev. de la tuberc. 1: 225, 1933.
7. Head, J. R.: J. THORACIC SURG. 6: 491, 1937.
8. Juabasic, D. M.: Wien klin. Wehnschr. 55: 481, 1942.
9. McIndoe, R. B., and Alexander, J.: Am. Rev. Tuberc. 29: 270, 1934.
10. McIndoe, R. B., Steele, J. D., and Alexander, J.: Am. Rev. Tuberc. 40: 243, 1939.
11. Ottaviano, Felix: Am. Rev. Tuberc. 41: 334, 1939.
Idem: Am. Rev. Tuberc. 43: 785, 1941.
12. Rogers, W. L.: Am. Rev. Tuberc. 24: 636, 1931.
13. Steele, J. D., Jr.: Am. Rev. Tuberc. 53: 184, 1946.

FIG. 3.

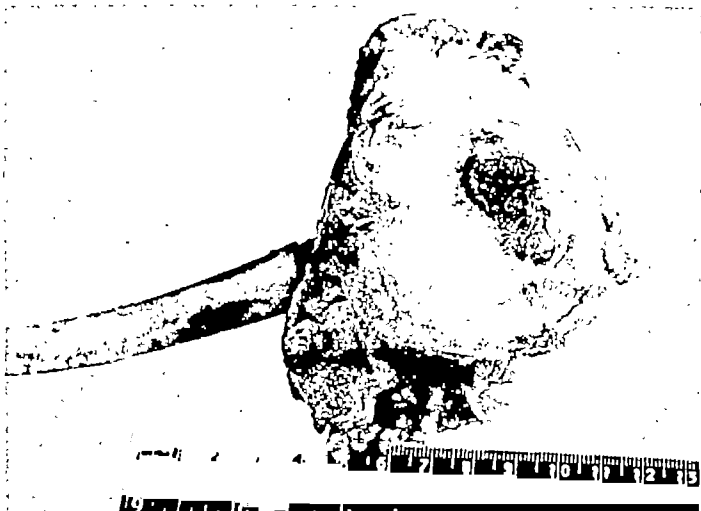


FIG. 4.

FIG. 3.—Tumor mass with involved lung tissue removed from left posterior mediastinum.

FIG. 4.—Same as FIG. 3, seen on cut section.

Neurosurgical exploration was decided upon as the first step in handling this patient. Accordingly, on Aug. 7, 1948, a spinal cord decompression was performed* by means of a left hemilaminectomy of the sixth and seventh thoracic vertebrae and a portion of an intraspinal, extradural tumor mass which was present in this area was removed. This tumor was reported by the pathologist to be a chondrosarcoma.

Following the neurosurgical operation the patient exhibited a marked improvement in general neurologic condition, and gradually regained the ability to walk. There was also a marked return of the sensory impairment previously noted. He continued unable to void, however, so that an indwelling catheter was utilized along with intermittent bladder irrigations and tidal drainage. Because of the patient's improvement and in spite of the invasive character of the tumor he was transferred to the Thoracic Surgical Service on Sept. 16, 1948, for further surgical therapy.



Fig. 1.

Fig. 1.—Roentgenogram showing round circumscribed tumor mass in the left paravertebral region.



Fig. 2.

Fig. 2.—Roentgenogram, left anterior oblique view, showing rounded sharply circumscribed tumor mass in the left paravertebral area with erosion of the vertebral end of the seventh rib.

A left thoracotomy was performed under general endotracheal anesthesia on Sept. 21, 1948. A posterolateral segment of the seventh rib was removed subperiosteally to about three inches from the angle of the rib. This rib appeared to be involved in the tumor mass as far laterally as the angle. Posterolateral segments of the eighth and sixth ribs were then removed in a similar manner as far as the rib necks posteriorly. The tumor could be seen to be infiltrating the apex of the lower lobe so that the pleural space was then entered through an incision in the bed of the seventh rib.

A segmental resection of the apex of the lower lobe was then accomplished leaving the involved lung and pleura attached to the mass. This procedure allowed mobilization and retraction of the lung so that the mediastinum could be visualized and approached.

The tumor was found to be a round, firm mass, about 10 cm. in diameter, apparently well contained except where it had involved the lung and seventh rib and at its points of attachment to the vertebral structures. It was apparently eroding and growing through

*By Dr. J. A. Mufson, the neurosurgical consultant.

types of tumors are apparent in a survey of the literature.¹⁻¹⁴ In a group of cases reviewed at the Mayo Clinic, Uhrich¹⁴ found that among 42 malignant mediastinal tumors, from which were excluded lymphomatous tumors and metastatic carcinomas, there were 6 chondrosarcomas. Heuer and Andrus¹¹ collected 14 cases of chondromatous tumors involving the mediastinum from the literature from 1926 to 1940; in addition, they reported 4 cases of this type from their own series of mediastinal tumors. Two of the tumors in their report involved the posterior mediastinum and one was an hourglass type of tumor causing cord compression within the spinal canal. Heuer¹⁰ had previously described a case reported by Elsberg of a chondrosarcoma of the posterior mediastinum involving the spinal cord.

The name of hourglass tumor has been applied to those tumors occurring along the spinal column and growing on both sides of an intervertebral foramen or through the space between two vertebral laminae. The tumor has derived its descriptive terminology from the constricted portion of the tumor extending through the intervertebral foramen or the interlaminar space. The group of these lesions in which the tumor we are reporting can be classed is that involving the posterior mediastinum as well as the spinal canal. The most common pathologic type of tumor occurring in this double location has been the neurofibroma,^{2, 10, 11, 12, 14} although a large variety of pathologic lesions have been reported as causing this type of tumor. Among the various histologic types reported have been the neuromas, fibromas, ganglioneuromas, lipomas, chondromas, fibrosarcomas, spongioblastomas, liomyomas, endotheliomas, neuroblastomas, and hemangioendotheliomas.

The specific origin of the tumor in the case herein reported is difficult to assess, but the abundance of ligaments, fascia, periosteum, and cartilage in the articular surfaces of the ribs and vertebrae in the posterior mediastinum provide sufficient possible sources of development of tumors of this type. The similarity of the chondrosarcomas to osteogenic sarcomas has also raised the question as to whether these tumors might not be osteogenic in origin. The significance of the previous trauma elicited in the history of this patient is probably doubtful, but it is interesting in view of the frequency with which previous trauma is commonly revealed in the history of patients developing osteogenic sarcomas in other parts of the body.

The histology of the chondrosarcoma in this case was characterized by an atypical chondral tissue with an abundant hyaline matrix invading bone and bone marrow. There were numerous areas of necrosis and cystic degeneration. The apparent paucity of myxomatous tissue has been the chief feature not coinciding with most of the pathologic descriptions of those tumors appearing in the literature.

The prognosis in this case is of course uncertain but the improvement gained in the first six months postoperatively has been encouraging.

SUMMARY

A case is reported of a 40-year-old white man presenting a chondrosarcoma of the posterior mediastinum producing an hourglass type of tumor with spinal

the lateral foramen between the sixth and seventh vertebrae, but it also seemed adherent to the sixth and eighth rib heads, the remaining lamina, pedicle, and a small lateral portion of the body of the sixth thoracic vertebra, and the intervertebral and posterior intercostal structures between the sixth and eighth thoracic vertebrae.

The tumor was removed en bloc excising the involved rib heads, vertebral lamina, pedicles, and portions of the vertebral body, and intervertebral and intercostal structures with the tumor. It was necessary to excise a portion of the dura to remove the tumor, and the pedicles and left lamina of the sixth and eighth vertebrae were resected to secure adequate visualization and removal of the tumor extending within the spinal canal. The dural fistula thus created was repaired by the use of Gelfoam film. The chest wall was then reconstructed in layers as far as possible with interrupted cotton sutures. The large pleurointercostal defect was covered by a sheet of Gelfoam film sutured to the edge of the remaining parietal pleura, and this was covered by the posterior chest wall and back muscles, and skin, and the wound was closed without drainage.

The pathologic description was as follows.* The gross specimen consisted of a roughly spherical mass measuring 10 cm. in its greatest diameter (Figs. 3 and 4). Over the surface was a smooth, shiny, gray-pink capsule similar to pleura. Attached to the external surface of this capsule was an area of mottled, gray-black pleura and lung substance. This capsule covered approximately one-half of the spherical mass. The opposite hemisphere was irregular in contour and in areas it was honeycombed in appearance. In areas there were pieces of dark red muscle protruding from the surface. Numerous tendinous bands coursed across the surface. Incorporated in this mass was a large portion of rib and several smaller segments of bone and ribs.

The tissue cut with marked resistance. The cut surface in portions was soft and semigelatinous in consistency. In other areas it was pink and gray and translucent in appearance.

The microscopic section showed cartilaginous tissue invading bone and bone marrow. There were some areas with necrotic cells while in others large oval-shaped, vesicular chondroblasts within lacunae were seen. The matrix appeared that of immature cartilage and there was an abundance of hyaline material.

The patient's postoperative course was satisfactory although complicated by a recurring partial hydropneumothorax which required aspiration of 200 to 400 c.c. of air and fluid on six occasions. Intensive physiotherapy begun before the operation was restarted on the day of operation directed at breathing exercises, weight-resistive exercises of the legs, and walking training. This therapy was continued daily up to his discharge from the hospital.

On Oct. 5, 1948, the indwelling catheter was removed and spontaneous normal micturition began that day. No significant residual urine was found on rechecking the bladder urine.

The patient demonstrated a gradual progressive improvement in walking and in the respiratory motions of the left chest. On Oct. 8, 1948, the roentgenograms of the chest showed complete re-aeration of the left lung with slight pleural thickening at the left base and no significant pneumothorax. He was discharged from the hospital on Nov. 15, 1948, complaining only of some residual soreness of the back, but as we learned later he was able to go deer hunting two days after he left the hospital.

The patient returned to this hospital for evaluation on Dec. 15, 1948, and examination revealed further progress in walking; his posture was normal, and the wound was well healed. Roentgenograms of the chest showed the postoperative changes incident to the rib and partial vertebral resections, but no other abnormalities were noted.

He returned again on March 14, 1949, for further follow-up study and was found to be in excellent health and able to get around well. Repeat roentgenograms of the chest were negative other than the changes incident to surgery mentioned previously.

DISCUSSION

The rarity of chondrosarcomas occurring in the posterior mediastinum and the even more infrequent reports of these lesions causing hourglass or dumbbell

*Joseph M. Lubitz, pathologist.

INLYING RUBBER TUBE AFTER ESOPHAGEAL ANASTOMOSIS

W. RALPH DEATON, JR., M.D., R. W. POSTLETHWAIT, M.D.,

H. H. BRADSHAW, M.D., AND ROBERT W. WILLIAMS, M.D.

WINSTON-SALEM, N. C.

THIS study was prompted by the death of a 78-year-old colored man on the third day after resection of a carcinoma of the middle third of the esophagus. No tube with intragastric suction had been employed; the vagotomized, intrathoracic stomach apparently gradually accumulated large quantities of fluid. Regurgitation and aspiration of this material resulted in the patient drowning in gastric secretions, as found at autopsy. Had intragastric suction been used, a favorable outcome might have resulted.

In the literature, varying opinions are found regarding the relative values or dangers of an inlying tube after esophagoesophageal and esophagogastric anastomosis. Those experienced in the treatment of congenital atresia of the esophagus with or without tracheoesophageal fistula generally oppose the use of a tube. Haight and Towsley¹ felt that the catheter increased pharyngeal secretions, that it tended to obstruct the esophagus at the anastomosis allowing accumulation of secretion with spill into the trachea, and that healing was more satisfactory if a foreign body was not present within the esophageal lumen. Humphreys² believed the tube stimulated sucking and increased oral secretions. Ladd and Swenson³ did not use a tube as this "diminishes the chance of respiratory infection and infection of the anastomosis." Gross⁴ omitted the tube because then "The esophagus can be given the best chance for healing during the early postoperative period." With reference to esophageal resection for carcinoma, Garlock⁵ stated, "The situation at the suture line is precarious enough without adding the danger of pressure necrosis from a Levin tube." Rankin and Hurt⁶ left a tube down four to five days, and Griswold⁷ for seventy-two hours. Nagel and Menke⁸ kept the tube down about five days "to prevent vomiting which would cause strain on the suture line." Thorek⁹ never hesitated to employ a tube as he believed "If the caliber of the tube over which the anastomosis is made is smaller than the stoma through which it passes, pressure is avoided and the blood supply is not impaired."

In order to study this problem, the following experiment was carried out.

METHOD

Mongrel dogs weighing 8 to 16 pounds were used. Thoracotomy on the left was performed under Nembutal anesthesia, with endotracheal intubation. The lower third of the esophagus was freed up, care being taken to preserve the vagus nerves and as much of the blood supply as possible. The esophagus was divided transversely. An anastomosis was carried out using two layers of interrupted 00000 silk sutures, one layer in the mucosa and submucosa, and the second layer in the muscular coats. The esophagus was handled carefully and the sutures placed meticulously. Before the chest was closed, an assistant passed a rubber tube down the esophagus into the stomach. After the operation was completed, the tube was wired to the lower molars on one side and the excess tube cut off. Most of the tubes were regurgitated and chewed off, so that the duration given in the results for the time the tube was in place indicates the time of the last examination when the tube was known to be intact. The exceptions were the three tubes removed at ninety-six hours.

From the Department of Surgery, Bowman Gray School of Medicine of Wake Forest College.

Received for publication April 22, 1949.

cord compression causing lower extremity paralysis. There was also invasion of the pleura, lung, ribs, and adjacent thoracic vertebrae.

The tumor was removed through a posterolateral thoracotomy following an initial laminectomy and spinal cord decompression, with immediate recovery and clinical improvement.

ADDENDUM

The patient was examined thoroughly on Dec. 16, 1949. He showed a remarkable clinical improvement and there was no evidence clinically or radiologically of a recurrence of the tumor.

REFERENCES

1. Blades, Brian: Relative Frequency and Site of Predilection of Intrathoracic Tumors, *Am. J. Surg.* 54: 139-148, 1941.
2. Blades, Brian: Mediastinal Tumors; Report of Cases Treated at Army Thoracic Surgery Centers in United States, *Ann. Surg.* 123: 749-765, 1946.
3. Freedlander, S. O.: Tumors of Mediastinum, *Ohio State M. J.* 38: 919-921, 1942.
4. Graham, E. A., Singer, J. J., and Ballou, H. C.: *Surgical Diseases of the Chest*, Philadelphia, 1935, Lea and Febiger, Chap. VI, pp. 189-269.
5. Harrington, S. W.: Surgical Treatment of Intrathoracic Tumors, *Arch. Surg.* 19: 1679-1725, 1929.
6. Harrington, S. W.: Intrathoracic New Growths; Results of Surgical Treatment in 24 Cases, *Surg., Gynec. & Obst.* 51: 647-666, 1930.
7. Harrington, S. W.: Diagnosis and Surgical Treatment of Anterior and Posterior Mediastinal Tumors; Report of Case of Posterior Mediastinal Tumor, *New York State J. Med.* 35: 1073-1080, 1935.
8. Harrington, S. W.: Value of Roentgen Rays in Diagnosis and Surgical Treatment of Extrapulmonary Intrathoracic Tumors, *Am. J. Roentgenol.* 33: 340-349, 1935.
9. Harrington, S. W.: Surgical Treatment in Sixteen Cases of Anterior Mediastinal Teratoid Tumors; One Complete Report of Case, *J. THORACIC SURG.* 7: 191-205, 1937.
10. Heuer, G. J.: So-Called Hour-Glass Tumors of Spine, *Arch. Surg.* 18: 935-981, 1929.
11. Heuer, G. J., and Andrus, W. DeW.: Surgery of Mediastinal Tumors, *Am. J. Surg.* 50: 143-224, 1940.
12. Kent, E. M., Blades, B., Valle, A. R., and Graham, E. A.: Intrathoracic Neurogenic Tumors, *J. THORACIC SURG.* 13: 116-161, 1944.
13. Naffziger, H. C., and Brown, H. A.: Hour-Glass Tumors of Spine, *Arch. Neurol. & Psychiat.* 29: 561-584, 1933.
14. Uhrich, E. C.: Primary Mediastinal Tumors; A Histopathologic Study, Thesis, Graduate School, University of Minnesota, (Jan.) 1947.

The dogs were allowed water for the first two days, or until the tube was out. Soft food, including a protein hydrolysate, was then given until the seventh or eighth day when they received the regular kennel rations.

With the variations as indicated in Table I, the esophagus was removed in six or ninety days for gross and microscopic study.

RESULTS

The results are recorded in Table I. Three dogs had large tubes (1 cm. external diameter), and all died on the second day after operation. Each had marked swallowing effort and profuse salivation in the same manner as the animals of Dragstedt and Mullenix¹⁰ which were subjected to acute esophageal obstruction, and died of aspiration pneumonia in one to four days.

The tube used in the other animals was a No. 14 French Levin tube. From the table, it is readily apparent that no interference with healing occurred as a result of the tube in so far as could be determined by gross and microscopic study.

DISCUSSION

In infants who have had an esophageal anastomosis, the objections to the use of a tube would seem valid, because of the possibility of increasing the chances of excessive oral secretions and consequent aspiration pneumonia. In the adult, however, this should be less prone to occur as the patient should be able to expectorate or swallow such secretion. Resections for carcinoma of the esophagus usually involve interruption of the vagus nerves, and the vagotomized stomach may be expected to retain considerable fluid. If the inlying rubber tube does not interfere with healing of the anastomosis, intragastric suction should be a valuable supplement in the postoperative care of these patients.

Admittedly, the solution of problems related to the esophagus of man may not be answered without reservation on the basis of experiments in dogs, since the esophagus does differ in dog and in man.

CONCLUSION

Under the conditions of this experiment, an inlying rubber tube, No. 14 French in size, does not interfere with the healing of an esophageal anastomosis in dogs.

REFERENCES

1. Haight, C., and Towsley, H. A.: Congenital Atresia of the Esophagus With Tracheo-esophageal Fistula, *Surg., Gynec. & Obst.* 76: 672-688, 1943.
2. Humphreys, G. H.: The Surgical Treatment of Congenital Atresia of the Esophagus, *Surgery* 15: 801-823, 1944.
3. Ladd, W. E., and Swenson, O.: Esophageal Atresia and Tracheo-esophageal Fistula, *Ann. Surg.* 125: 23-40, 1947.
4. Gross, R. E.: Treatment of Short Stricture of the Esophagus by Partial Esophagectomy and End-to-end Esophageal Reconstruction, *Surgery* 23: 735-744, 1948.
5. Garlock, J. H.: The Surgical Treatment of Carcinoma of the Thoracic Esophagus, Presented at Clinical Congress of American College of Surgeons, Philadelphia, October, 1939.
6. Rankin, F. W., and Hurt, L. E.: Intrathoracic Esophago-gastric Anastomosis for Carcinoma of the Lower Esophagus and Cardiac End of the Stomach, *Kentucky M. J.* 46: 188-193, 1948.
7. Griswold, R. D.: Transthoracic Gastrectomy for Unusual Lesions of the Stomach, *Ann. Surg.* 121: 600-619, 1945.
8. Nagel, G. W., and Menke, J. F.: Transthoracic Operations for Neoplasms of the Esophagus and Stomach, *Surg., Gynec. & Obst.* 83: 657-666, 1946.
9. Thorek, P.: Surgical Treatment for Carcinoma of the Esophagus, *Illinois M. J.* 92: 329-337, 1947.
10. Dragstedt, C. A., and Mullenix, R. B.: Experimental Esophageal Obstruction, *Arch. Surg.* 24: 162-167, 1932.

TABLE I. SUMMARY OF RESULTS OF HEALING IN ESOPHAGEAL ANASTOMOSIS WITH INTACT RUBBER TUBE

NUMBER	HOURS TUBE IN PLACE	TIME- OPERATION TO AUTOPSY (DAYS)	GROSS FINDINGS	MICROSCOPIC FINDINGS			COMMENT
				MUCOSA	SUBMUCOSA	MUSCULAR	
1	No tube	6	Slight mucosal separation	Small ulcer	Moderate acute inflammation	Healing well	
2	No tube	7	Healing well*	Healing well	Minimal chronic inflammation	Healing well	
3	48†	2	Healing well		Not studied†		Died, pneumonia
4	48†	2	Healing well	Small ulcer	Moderate chronic inflammation	Moderate chronic inflammation	Died, pneumonia
5	48†	2	Healing well		Not studied†		Died, pneumonia
6	24	6	Healing well	Healing well	Moderate chronic inflammation	Healing well	
7	24	6	Healing well	Healing well	Healing well	Healing well	
8	72	9	Healing well	Healing well	Healing well	Healing well	
9	96	6	Healing well	Small ulcer			
10	96	6	Healing well	Healing well	Moderate chronic inflammation	Healing well	
11	48	42	Well healed*		Healing well	Slight chronic inflammation	
12	No tube	90	Well healed	Well healed	Well healed	Well healed	Died, pneumonia
13	24	90	Well healed	Well healed	Well healed	Well healed	No stricture
14	24	90	Well healed	Well healed	Well healed	Well healed	No stricture
15	30	90	Well healed	Well healed	Well healed	Well healed	No stricture
16	72	90	Well healed	Well healed	Well healed	Well healed	No stricture
17	96	90	Well healed	Well healed	Slight chronic inflammation	Well healed	No stricture

*Healing well indicates fibroblastic proliferation; well healed indicates mature scar tissue.

†Large tube.

‡Too autolyzed.

12. **Experimental Attempts at the Surgical Relief of Aortic Stenosis**
Charles P. Bailey, Robert P. Glover, Thomas J. E. O'Neill (by invitation), and Hector P. Redondo Ramirez (by invitation)
13. **The Experimental Creation and Closure of Auricular Septal Defects**
Henry Swan, George Maresk (by invitation), Marvin E. Johnson, and George Warner
14. **A Physiological Evaluation of Vagus Section for Bronchial Asthma**
Karl P. Klassen, Douglas R. Morton (by invitation), and George M. Curtis
15. **Therapeutic Status of Pulmonary Autonomic Nerve Surgery**
Osler A. Abbott, William A. Hopkins (by invitation), and Paul H. Guilfoill
16. **The Surgical Treatment of Intractable Asthma**
Brian Blades, Edward J. Beattie, Jr., and William S. Elias (by invitation)

Monday Afternoon, April 17, 1950

2:00 P.M. EXECUTIVE SESSION

3:00 P.M. SCIENTIFIC SESSION

Address of the President—Edward J. O'Brien

17. **The Experimental Use of Homogenous Tracheal Transplants**
Truxton D. Jackson (by invitation), Edward J. O'Brien, William Tuttle, and John Meyer (by invitation)
18. **Resection of the Trachea and Bronchi—An Experimental Study**
Max G. Carter (by invitation) and John W. Strieder
19. **Further Experiences With Dermal Grafts for Healed Tuberculous Stenosis of the Bronchi and Trachea**
Paul W. Gebauer

Tuesday Morning, April 18, 1950

9:00 A.M. SCIENTIFIC SESSION

20. **Congenital Atresia of the Esophagus With or Without Tracheo-esophageal Fistula**
Willis J. Potts
21. **Reconstruction of Esophagus**
Ross Robertson and S. T. R. Sarjeant (by invitation)
22. **Use of the Burns' Valve as a Simple Respirator for Intrathoracic Surgery in the Dog**
Joseph T. Dameron and David G. Greene (by invitation)
23. **The Development of and Certain Clinical Applications for a Portable Mass Spectrometer**
Fletcher A. Miller, A. O. C. Nier, Allan Homingway, Ralph T. Knight, E. B. Brown (by invitation), and Richard L. Varco
24. **The Surgical Treatment of Giant Emphysematous Blebs and Pulmonary Tension Cysts**
David J. Dugan and Paul C. Samson
25. **Experimental Tuberculosis. Effects of Ligation of Pulmonary Arteries on Tuberculosis in Monkeys**
H. William Scott, Jr. (by invitation), C. Rollins Hanlon, and Byron J. Olson (by invitation)

Tuesday Afternoon, April 18, 1950

2:00 P.M. SCIENTIFIC SESSION

26. **Pulmonary Function Before and After Extrapleural Pneumothorax**
Edward A. Graessler (by invitation), and John W. Strieder

THE AMERICAN ASSOCIATION FOR THORACIC SURGERY

THIRTIETH ANNUAL MEETING

APRIL 15, 16, 17, AND 18, 1950, HOTEL COSMOPOLITAN
DENVER, COLO.

PROGRAM

Saturday Morning, April 15, 1950

9:00 A.M. BUSINESS MEETING

9:30 A.M. SCIENTIFIC SESSION

1. **Intrathoracic Neurogenic Tumors**

J. T. Godwin (by invitation), W. L. Watson, J. Pool, and W. Cahan
(by invitation)

2. **Tumors of the Thymus**

W. D. Seybold, John R. McDonald, O. T. Clagett, and C. A. Good
(by invitation)

3. **Treatment of Carcinoma of the Lung Associated With Malignant
Tumors Primary in Other Sites; Report of Eight Cases**

William G. Cahan (by invitation), Frank S. Butler (by invitation),
William L. Watson, and John L. Pool

4. **The Surgical Management of Carcinoma of the Lung**

E. D. Churchill, R. H. Sweet, L. Soutter, and J. G. Scannell

5. **A Study of Pulmonary Hemodynamics During Pneumonectomy**

Harvey J. Mendelsohn (by invitation), Henry A. Zimmerman, and
Arthur Adelman

Saturday Afternoon, April 15, 1950

2:00 P.M. SCIENTIFIC SESSION

6. **Conservative Management of Empyema Following Total Pneumonec-
tomy**

Edward M. Kent

7. **The Utilization of Streptokinase-Streptodornase in: (1) A Patient
With Hemopneumothorax and (2) a Patient With Postpneumonectomy
Sanguineous Coagulum**

C. Thomas Read (by invitation), and Frank B. Berry

8. **Pulmonary Paraffinoma (Lipoid Pneumonia), A Critical Study**

Thomas H. Burford and Ralph Berg (by invitation)

9. **Surgery in Pulmonary Coccidioidomycosis—Preliminary Report of
Twenty-five Cases**

Bert H. Cotton and J. W. Birsner (by invitation)

10. **Pectus Excavatum—Management of a Case With Severe Cardiopulmo-
nary Disability With Pre- and Postoperative Angiocardiographic Studies**

Ralph A. Dorner, Philip G. Keil (by invitation), and Donald J.
Schissel (by invitation)

7:00 P.M. COCKTAIL PARTY—HOTEL COSMOPOLITAN

8:00 P.M. BANQUET—HOTEL COSMOPOLITAN
DANCING

Monday Morning, April 17, 1950

9:00 A.M. SCIENTIFIC SESSION

11. **Studies With Arteriovenous Fistulas. I. Response of the Normally
Innervated and Denervated Heart to Occlusion of the Fistula**

H. B. Shumacker, Jr., L. W. Freeman (by invitation), and Leo
Radigan (by invitation)

The Journal of Thoracic Surgery

VOL. 19

MAY, 1950

No. 5

Original Communications

PULMONARY RESECTION FOR METASTATIC MALIGNANT LESIONS

HAWLEY H. SEILER, M.D.,* O. THERON CLAGETT, M.D.,† AND

JOHN R. McDONALD, M.D.‡

ROCHESTER, MINN.

IT IS not surprising that with the increased incidence, in recent years, of pulmonary resection for bronchiogenic carcinoma, attention should sooner or later be directed toward surgical excision of metastatic lesions. Recently Alexander and Haight¹ and Effler and Blades² have reported series of pulmonary resections for solitary metastatic lesions and included reviews of the literature. Prior to these reviews, only a few isolated case reports had appeared in the literature. About two years ago, a study of this subject was started at the Mayo Clinic.³ This study included a review of the literature dealing with pulmonary metastatic phenomena and with resection for pulmonary metastatic lesions, together with a review of 10 cases in which resection had been performed at the clinic since 1941. The object of this study was to review the results obtained in all reported cases, including our own, in an effort to evaluate the worth of this procedure and to determine its place in modern thoracic surgery. Data on a total of 62 cases were assembled, including the 10 Mayo Clinic cases.

REVIEW OF THE LITERATURE

It is indeed surprising how far back attempts at surgical removal of pulmonary tissue for secondary malignant lesions can be traced. In 1898, there appeared in the German literature, an extremely interesting paper by Gerulanos,⁴ in which he reported removing a portion of lung of a 15-year-old boy for a small round-cell sarcoma which had invaded from the chest wall. The patient died. Gerulanos further reviewed the data on 38 previously reported cases of chest wall tumors which had been subjected to surgical attack, and in 5 instances

*Read at the Twenty-ninth Annual Meeting of The American Association for Thoracic Surgery, New Orleans, La., March 29-31, 1948.

†Abridgment of thesis submitted by Dr. Seiler to the Faculty of the Graduate School of the University of Minnesota in partial fulfillment of the requirements for the degree of Master of Science in Surgery.

‡Fellow in Surgery, Mayo Foundation.

§Division of Surgery.

||Division of Surgical Pathology, Mayo Clinic.

27. Summary of Pulmonary Tuberculomas, Pathogenesis, Diagnosis and Management

Gordon J. Culver (by invitation), Joseph P. Concannon (by invitation), and Joseph E. Macmanus

28. One-Stage Thoracoplasty for Pulmonary Tuberculosis

R. C. Laird and C. E. Lindenfield (by invitation)

29. Further Experiences With Segmental Resection in Pulmonary Tuberculosis

J. Maxwell Chamberlain and Robert Klopstock (by invitation)

30. Resection in the Treatment of Pulmonary Tuberculosis

William M. Tuttle, E. J. O'Brien, J. Claude Day, Foster Hampton, Jr. (by invitation), and Truxton L. Jackson, M.D.

31. Pulmonary Resection in Pulmonary Tuberculosis

A. Himmelstein, Frank B. Berry, and C. T. Read (by invitation)

PAPERS TO BE READ BY TITLE

1. Temporary Extrafascial Lucite Plombage and Thoracoplasty

Elliott Michelson, George W. Wright, and Bernard J. Ryan (by invitation)

2. Studies in Esophageal Replacement—Influence of Staging of Ligation of Mesenteric Vessels Upon Collateral Blood Supply of Intestinal Segments Freed for Transplantation

H. B. Shumacker, Jr., and J. S. Battersby (by invitation)

3. Further Experiences With the Surgical Treatment of Mitral Stenosis by Commissurotomy

Thomas J. E. O'Neill (by invitation), Robert P. Glover, Charles P. Bailey, and Hector P. Redondo Ramirez (by invitation)

4. Bronchography as an Aid in Surgical Planning in the Treatment of Pulmonary Tuberculosis

Joseph Gordon

patient was subsequently reported as being alive and well twelve years after the pulmonary operation.¹²

Experience with this case and with subsequent ones led Churchill¹³ to comment, in 1940, as follows: "The status of pulmonary resection for known metastatic tumors of the lung is admittedly one of experimentation. In general, it may be taken under consideration when a solitary metastasis of a tumor of relatively low-grade malignancy appears in the lung after complete eradication of the primary growth. It is also to be considered in cases of hypernephroma with a solitary pulmonary metastasis."

In discussing all pulmonary resections for malignant disease done by him in the ten-year period prior to 1940, Churchill mentioned 5 more resections for metastatic tumors in addition to the case previously reported with Barney.

Following these early cases, each successive year has seen the appearance in the literature of additional reports of pulmonary resection for metastatic malignant tumors. In 1941, Cotton¹⁴ mentioned a case of pneumonectomy performed for adenocarcinoma metastatic from the cecum, and Raine¹⁵ reported a case of pneumonectomy for metastatic carcinoma originating in the sigmoid. In 1942, Freedlander and Greenfield¹⁶ reported 2 cases, and the following year saw reports of 1 case each by King and Castleman¹⁷ and by Brezina and Lindskog.¹⁸ The primary sites of these 4 tumors were ovary, thyroid, stomach, and uterus. Deming and Lindskog¹⁸ described, in 1944, a case of pneumonectomy for carcinoma metastatic from the bladder and urethra, and in the same year Blalock²⁰ mentioned having done a pneumonectomy for a metastatic lesion from a primary carcinoma of the rectum. In 1945, Tinney and McDonald²¹ referred to a case of pneumonectomy performed at the Mayo Clinic for metastatic hypernephroma. This case is included among the report of 10 cases to be presented later in this paper.

The late Tudor Edwards,²² in an article appearing just before his death in which he summarized his experience over the years with bronchiogenic carcinoma, made reference to the problem of pulmonary resection in secondary malignant disease. He had performed resection in 4 such cases, and 1 of the patients was alive eighteen years later. This is apparently the longest recorded survival following resection of a pulmonary metastatic lesion. Subtotal lobectomy was performed in this case, and the primary tumor was a sarcoma of the fibula. Unfortunately, no further details of the case were given. Mandeville²³ reviewed this subject and added 4 cases of his own, and Aub and Mallory added another case of lobectomy for metastatic hypernephroma (Cabot case 33,012).²⁴ Overholt and Langer,²⁵ in their paper dealing with the technique of pulmonary segmental resection, referred to 1 case of resection performed for metastatic myxosarcoma. Maier and Taylor²⁶ described a case of metastatic chorionepithelioma treated by lobectomy, and Rienhoff,²⁷ in discussing the present status of the surgical treatment of carcinoma of the lung, made reference to 4 cases of pulmonary extirpation for metastatic malignant disease. Two of these were personal cases and 2 cases were quoted from Blalock.

portions of lung were also removed for secondary malignant lesions. In 4 of these 5 cases the patients survived operation; 3 were reported as cured and 1 died of recurrence of sarcoma. The earliest of these 5 case reports was published in 1855 and the surgeon was a Frenchman by the name of Sedillot.⁵ Of course, these early cases all represented direct spread of cancer from the chest wall rather than discrete metastatic growths.

In 1923, Meyer⁶ reported the case of a left pneumonectomy performed on a boy, aged 5 years, the patient dying ten hours postoperatively. Microscopically, the lung showed lymphosarcoma and bronchiectasis. Whether or not this lesion was primary or secondary in the lung we do not know inasmuch as only partial necropsy was performed.

Perhaps the first proved and complete case history of pulmonary resection for a metastatic malignant lesion was that of Diviš⁷ reported in 1927. The patient was a 42-year-old woman who had undergone ten operations in the previous thirty-five years for the removal of a recurring, slowly growing sarcoma of the left breast. A solitary tumor developed in the lower lobe of the right lung, and this was removed surgically and shown to be identical microscopically to the sarcoma of the breast. It is interesting to note that the author believed, before operation, that this lesion was metastatic, and based his decision to operate on the fact that the primary tumor was apparently a slowly growing one; the metastatic lesion appeared to be small, well defined, and solitary, and the patient was in excellent general health. At the time of the report, the patient had been well for six months with no evidence of recurrence.

Torek⁸ reported an interesting case in 1930. His patient was a 42-year-old woman from whom he removed a metastatic adeno-acanthoma of the lung three years after hysterectomy for the primary tumor. The correct diagnosis was made only subsequent to operation by the pathologist. However, in commenting upon this case, Torek stated that having had such an experience, he would not be too dogmatic in refusing to remove such a metastatic lesion in the future. The patient was relieved of symptoms and was well at the time of the report.

In 1933, Lilienthal⁹ reported the case of a pneumonectomy performed for a tumor of the left lung in a tuberculous patient, which proved to be metastatic from sarcoma of the uterus. The patient died on the fourth post-operative day and the primary lesion was discovered at necropsy.

Röpke¹⁰ reported 2 cases of lobectomy for metastatic carcinoma in 1937. The primary cancers in these 2 instances were carcinoma of the breast and carcinoma of the ovary, respectively. The first patient died several months later with cerebral metastasis while the second was alive and well three and one-half years later.

In 1939, appeared the outstanding report of Barney and Churchill.¹¹ Their patient had been subjected to nephrectomy for adenocarcinoma in April, 1932, and pulmonary resection was performed in July, 1933, to remove a metastatic tumor. Actually, the metastatic lesion had been seen roentgenographically some time before discovery and removal of the renal tumor. This

This patient is of interest because she survived almost seven years after pulmonary resection and because the metastatic lesion was diagnosed bronchoscopically.

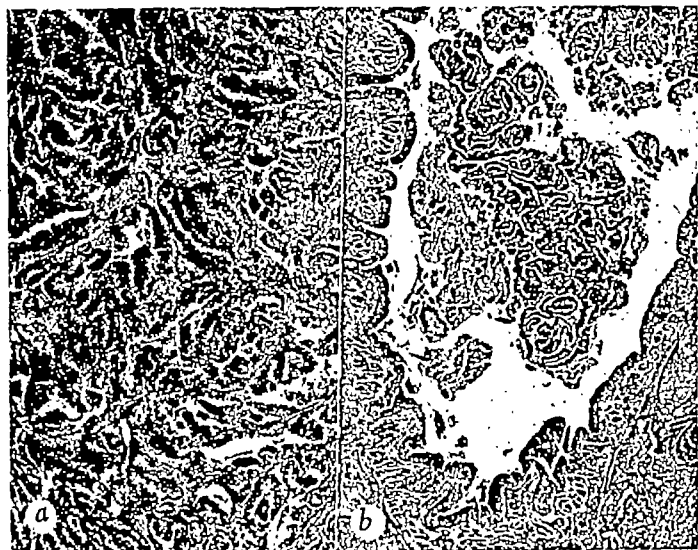


Fig. 2 (Case 1).—*a*, Primary hypernephroma of the kidney, showing characteristic grouping of neoplastic cells (hematoxylin and eosin $\times 25$). *b*, Metastatic hypernephroma of the left lung with a pattern similar to that of the primary tumor. The section shows tumor tissue within a major bronchus (hematoxylin and eosin $\times 25$).

CASE 2.—A 38-year-old housewife was seen in March, 1943, with two major pathologic findings: one, an obstructing, annular carcinoma of the rectum, and the other, roentgenographic evidence of a mass in the lower portion of the right hilar region. Biopsy of both lesions showed them to be identical grade 1 adenocarcinomas. A preliminary colostomy was done, and during the interval before proceeding with resection of the bowel, a right pneumonectomy was performed. This was followed in several months by anterior resection of the pelvic colon with end-to-end anastomosis. The patient remained well for about one and one-half years at which time she returned with obstruction of the bowel and abdominal carcinomatosis. She died two months later.

CASE 3.—A 40-year-old white barber was seen in August, 1943, with a spherical mass in the apex of the left lung. He had complained of hemoptysis for fourteen months. Vague abdominal symptoms in the form of dyspepsia and loose stools were occasionally noted also. Pneumonectomy was performed, and the specimen histologically proved to be an anaplastic, grade 4 carcinoma showing feeble attempts at gland formation. On the twenty-first post-operative day, abdominal pain and rigidity developed, and evidence of a perforated viscus and peritonitis soon became evident. General toxicity and shock rapidly developed and in forty-eight hours the patient was dead. Post-mortem examination revealed a tumor of the jejunum which had perforated with resulting peritonitis and death. Microscopically, the tumor closely resembled the lesion in the lung and was thought to be the primary tumor in this case, with the pulmonary lesion being a metastatic one.

In August, 1947, appeared Alexander and Haight's¹ article in which 24 cases were reported. Six of these were personal cases while data on the remaining 18 were assembled from the literature and by direct inquiry from other thoracic surgeons. Some of these cases have already been mentioned but others not previously referred to include 6 cases by Sweet,²⁸ and 1 each by Dolley,²⁹ Carr,³⁰ and Samson.³¹ Four Mayo Clinic cases were also included by these authors.

Recently, Effler and Blades² wrote on this subject and reported 3 cases. They also discussed some of the cases which had been reported in the literature and described 1 by Flick³² which had not been previously reported.

REPORT OF CASES

CASE 1.—A 39-year-old housewife came to the clinic complaining of hematuria of nine months' duration. A pyelogram on admission disclosed a right renal tumor for which nephrectomy was performed in July, 1938. The pathologic examination proved this tumor to be a grade 1 hypernephroma with involvement of the renal vein by a thrombus of tumor tissue. A course of roentgen therapy was given postoperatively, and the patient remained well for several years. In July, 1940, the patient complained of pulmonary symptoms, namely, cough, chest pain, hemoptysis, and dyspnea, and a roentgenogram of the chest revealed an infiltrating lesion involving the left hilus and upper lobe (Fig. 1). Bronchoscopy revealed a



Fig. 1 (Case 1).—Infiltration of the left hilus and left upper lobe by metastatic hypernephroma.

tumor projecting into the left main bronchus, which on biopsy was reported as only inflammatory tissue. However, metastatic hypernephroma was suspected and roentgen therapy was given. In April, 1941, bronchoscopic biopsy was positive for metastatic hypernephroma, and a left pneumonectomy was performed in August, 1941.

The patient remained well for a number of years. In February, 1947, she complained of cough and occasional hemoptysis, and a roentgenogram of the chest revealed a pathologic process in the right lower pulmonary field. Symptoms progressed and in January, 1948, the patient coughed up a piece of tissue which proved histologically to be metastatic hypernephroma (Fig. 2). She finally died in May, 1948.

This case is a good example of what Willis²³ referred to as a "latent primary neoplasm" in which the first indication of cancer is manifest by the appearance of a "precocious metastasis."

CASE 4.—A 25-year-old physician was seen in May, 1944, fourteen months subsequent to left orchidectomy for teratoma. There were no pulmonary symptoms, but a roentgenogram of the chest revealed a lobulated shadow in the lower right pulmonary field. The lesion failed to respond to irradiation therapy, and consequently a right lower lobectomy was performed. As suspected, the extirpated portion of lung contained metastatic teratoma. This patient survived twenty-one months following pulmonary resection and died as the result of further intrapulmonary metastasis.

CASE 5.—In August, 1941, a 46-year-old man was subjected to posterior resection of the rectum following preliminary loop colostomy for grade 2, ulcerating adenocarcinoma. The patient remained well for four years. At the time of the periodic re-examination in October, 1945, a solitary nodule in the right upper lobe was seen on the roentgenogram (Fig. 3). There were no symptoms. Right upper lobectomy was performed in November, 1945, and the excised lesion presented a histologic pattern identical to that of the previously removed rectal carcinoma (Fig. 4). Unfortunately, cerebral metastatic lesions developed eight months later, and the patient died about a year after lobectomy.

CASE 6.—A 34-year-old man underwent amputation of the right leg in January, 1942, for a recurrent, grade 1 chondrosarcoma with areas of osteogenic sarcoma of the os calcis. The tumor had been locally excised on four previous occasions. After amputation he remained well for about four years, and then returned to the clinic complaining of productive cough, dyspnea, and a recent acute respiratory infection. A roentgenogram revealed a mass in the right upper pulmonary field that contained areas of calcification. Pulmonary resection was advised and a right upper lobectomy performed. It was impossible to remove all of the tumor at the time of operation, however, owing to mediastinal and chest wall involvement, so that the procedure must be classed as a frankly palliative one. Pathologic examination showed this tumor to be an osteogenic sarcoma also. The patient survived only several months after operation.

CASE 7.—A 23-year-old housewife was first seen at the clinic in 1924, at which time electrosurgical excision of an adenocarcinoma, mixed tumor type, of the alveolar process was done. The tumor recurred locally in 1927, and was again removed by destroying the entire site with diathermy. Postoperative radium therapy was used each time.

The patient remained well for many years and returned in April, 1946, at which time a well-circumscribed, nodular tumor was found in the right midpulmonary field (Fig. 5). Pneumonectomy was performed and the extirpated lesion was demonstrated to be a metastatic adenocarcinoma, mixed tumor type (Fig. 6). Some months later the patient returned with further peripheral metastatic growths in the right breast, the scalp, and the left groin. These were proved by biopsy. She died one year after pulmonary resection owing to further metastatic spread, principally intra-abdominal.

CASE 8.—A 28-year-old farmer was seen at the clinic in October, 1946, with a pulmonary tumor obstructing the bronchi of the right middle and lower lobes and producing partial atelectasis. History disclosed orchidectomy three years before for a grade 4 malignant tumor. Biopsy of the lesion in the lung showed it to be a grade 4 squamous cell epithelioma. A right pneumonectomy was performed with uneventful recovery. Histologic comparison of the two tumors showed them to be similar, both lesions proving to be grade 4 mixed squamous-cell epithelioma and adenocarcinoma. A two months' follow-up indicated recurrence of the pulmonary lesion, and the patient lived only four months after resection.

CASE 9.—A 20-year-old girl presented two lesions: a recurrent fibrosarcoma of the lower part of the right leg, and a well-circumscribed roentgenographic shadow in the right lower

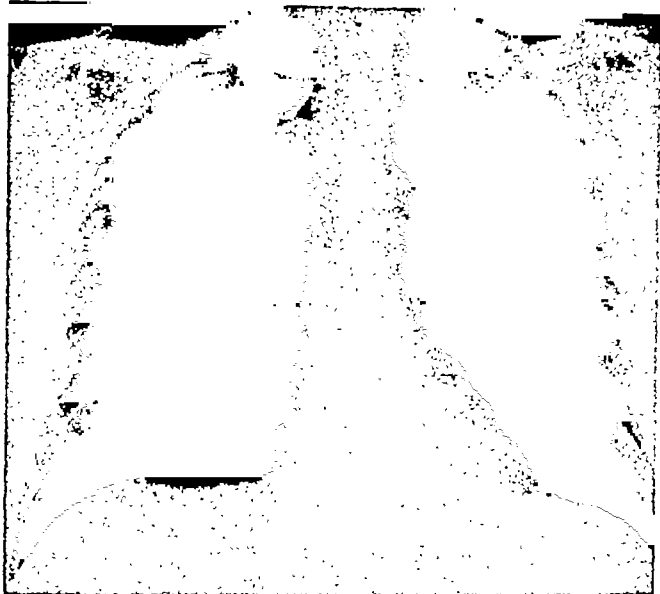


Fig. 3 (Case 5).—Metastatic lesion in the right upper lobe close to the mediastinum.

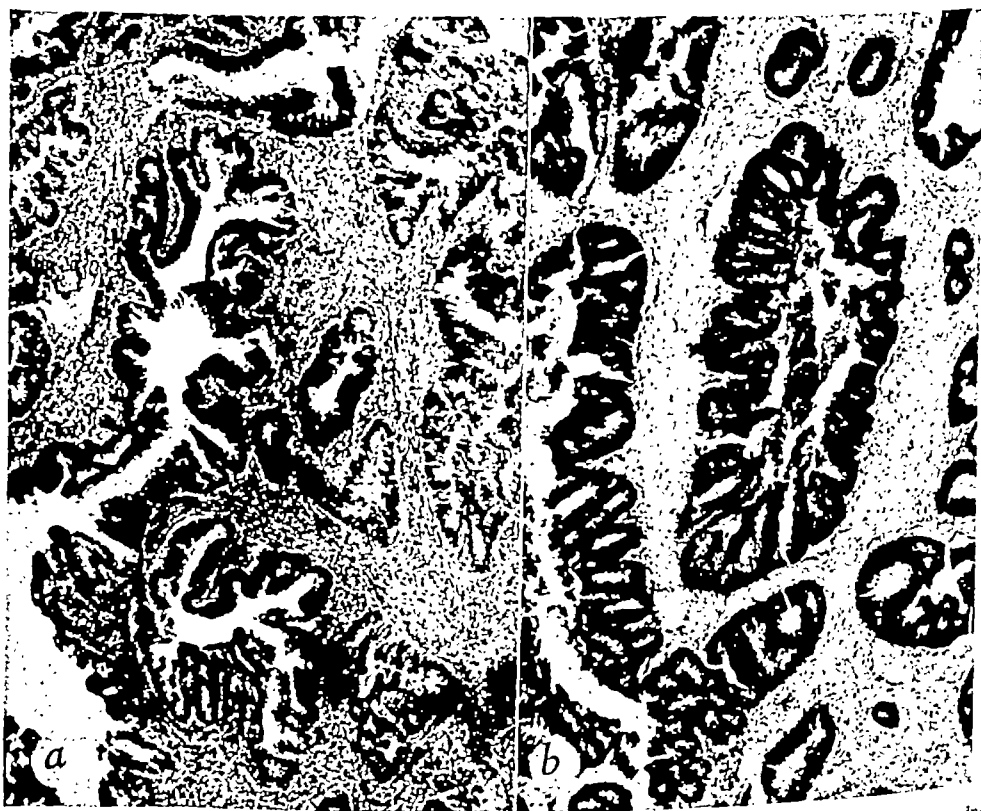


Fig. 4 (Case 5).—*a*, Primary grade 2 adenocarcinoma of the rectum with papillary infoldings (hematoxylin and eosin $\times 55$). *b*, Metastatic grade 2 adenocarcinoma of the lung similar in pattern to primary rectal tumor (hematoxylin and eosin $\times 55$).

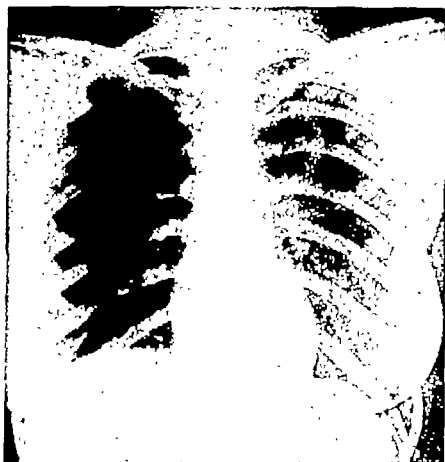


Fig. 7 (Case 9).—Well-circumscribed, metastatic fibrosarcoma peripherally situated in the right lower pulmonary field.

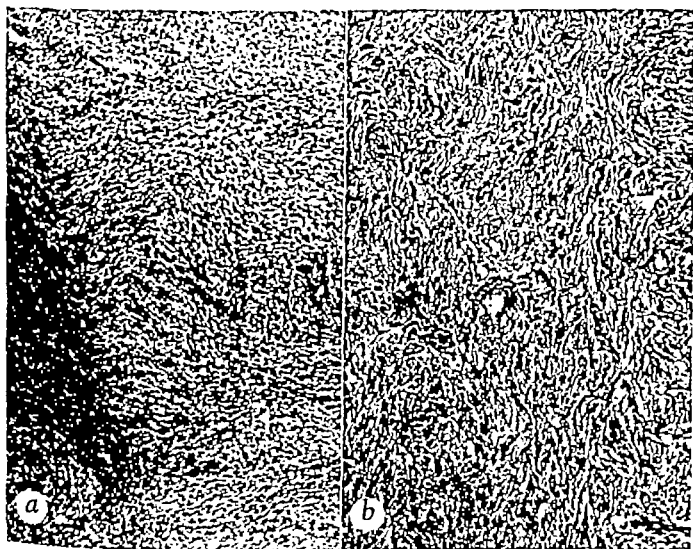


Fig. 8 (Case 9).—a, Primary grade 1 fibrosarcoma of the right leg; spindle-shaped cells are compactly arranged (hematoxylin and eosin $\times 85$). b, Metastatic grade 1 fibrosarcoma of the lung; the pattern of interlacing bundles is similar to that in a (hematoxylin and eosin $\times 85$).



Fig. 5 (Case 7).—Nodular, metastatic tumor in the right lung.

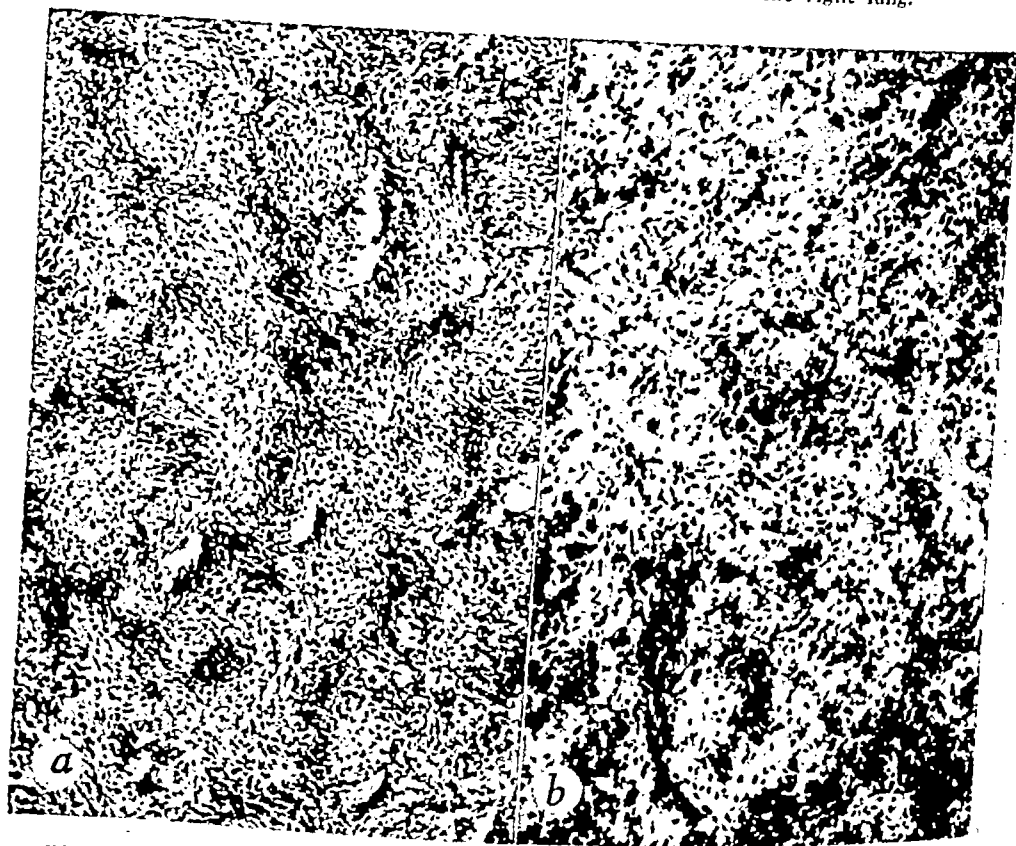


Fig. 6 (Case 7).—*a*, Primary adenocarcinoma of the jaw, mixed tumor type. Cartilage formation can be seen (hematoxylin and eosin $\times 85$). *b*, Metastatic adenocarcinoma of the lung, mixed tumor type, with epithelial cords between areas of cartilage (hematoxylin and eosin $\times 85$).

formed, and the tumor of the leg was again widely excised. Pathologic examination of the two lesions showed them to be identical in every respect (Fig. 8). The patient has been heard from a number of times and has remained well.

CASE 10.—A 51-year-old housewife was seen in January, 1947, when a well-circumscribed tumor was visible roentgenographically in the base of the right lung (Fig. 9). She had had two previous operations for cancer, the first being enucleation of the right eye in 1936, because of melano-epithelioma, and the second being right oophorectomy and hysterectomy in 1943, for adenocarcinoma of the ovary. Bronchoscopic biopsy of the pulmonary tumor showed it to be adenocarcinoma, and a right lower lobectomy was performed. Histologic comparison of the removed pulmonary tumor and the original carcinoma of the ovary showed them both to be grade 4, papillary adenocarcinoma (Fig. 10). This patient has remained well with no evidence of recurrence.

ANALYSIS OF DATA ON MAYO CLINIC CASES

Type of Primary Tumor.—Of the 10 cases reported herein, 5 represented carcinomas, 3 mixed tumors, and 2 sarcomas. The mixed tumors were predominantly carcinomatous, however, and have been included as such in Table I.

TABLE I. TYPE OF PRIMARY TUMOR (MAYO CLINIC CASES) FOR WHICH PULMONARY RESECTION OF THE METASTATIC LESION WAS CARRIED OUT

TYPE OF PRIMARY TUMOR	CASES
Carcinoma	8
Mixed tumor	3
(adenocarcinoma of jaw, mixed tumor type, 1; mixed squamous-cell epithelioma and adenocarcinoma of testis, 1; teratoma of testis, 1)	
Adenocarcinoma of rectum	2
Adenocarcinoma of jejunum	1
Adenocarcinoma of ovary	1
Hypernephroma	1
Sarcoma	2
Osteogenic sarcoma of os calcis	1
Fibrosarcoma of leg	1

Symptoms.—A review of the symptoms elicited from the various patients at the time of discovery of the pulmonary lesion is of relatively little value as an aid to diagnosis. Three patients were asymptomatic. Cough and dyspnea were the two most common symptoms, each being complained of by 4 patients. In 3 patients hemoptysis had been experienced, and 3 patients complained

TABLE II. SURVIVAL PERIOD FOLLOWING PULMONARY RESECTION FOR A METASTATIC MALIGNANT LESION (MAYO CLINIC CASES)

CASE	SURVIVAL (MONTHS)	PRESENT STATUS OF PATIENT
1	81	Dead
2	24	Dead
3	23 days	Dead
4	21	Dead
5	12	Dead
6	3	Dead
7	12	Dead
8	4	Dead
9	28	Alive and well
10	24	Alive and well

pulmonary field (Fig. 7). The tumor of the leg had been removed in 1935, and again in 1939, and review of the histologic sections showed it to be a slowly growing, grade 1 fibrosarcoma. The patient steadfastly refused amputation. In October, 1946, right lower lobectomy was per-

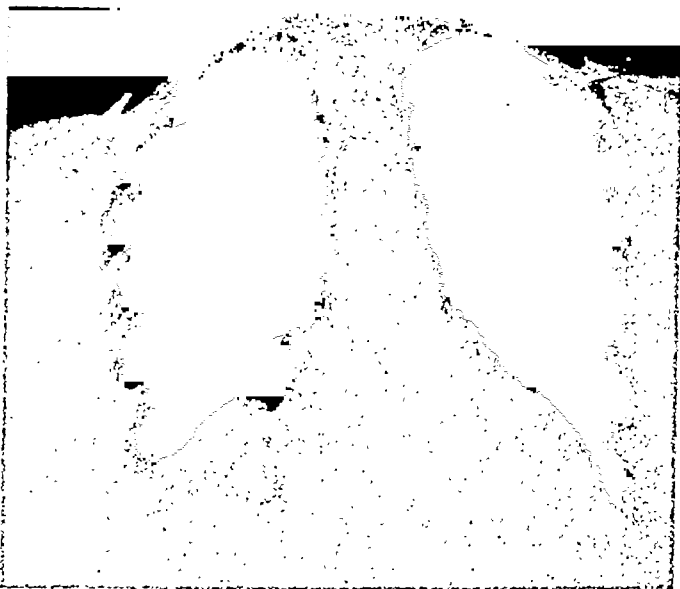


Fig. 9 (Case 10).—Metastatic tumor in the right lower pulmonary field.

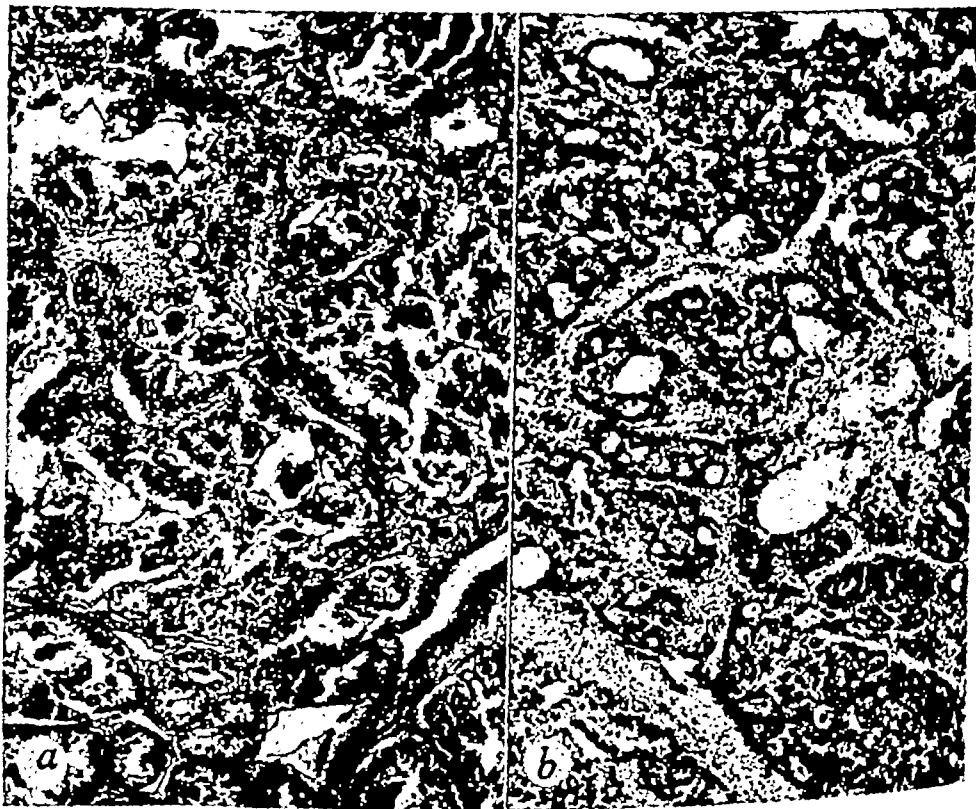


Fig. 10 (Case 10).—*a*, Primary ovarian tumor, a grade 4, papillary colloid adenocarcinoma (hematoxylin and eosin $\times 50$). *b*, Metastatic tumor of the lung showing similar grade 4, papillary, colloid adenocarcinoma (hematoxylin and eosin $\times 55$).

Year	Author (Case #)	Location, tissue	Not stated 60 to 12 (%)	00 to 12 (%)	Subtotal lobectomy Pneumonectomy	No data No data	216	8½ (f) 12 (f) "Several", No data	Well Death at operation 8½
1946	Edwards (Case 4)	Sarcoma, thibula	Not stated	00 to 12 (%)	Subtotal lobectomy	No data			
1946	Manderville ¹ (Case 1)	Fibrosarcoma, arm	48	72	Pneumonectomy	No data			
1946	Manderville (Case 2)	Granulosa-cell tumor, ovary	48	48	Pneumonectomy	No data			
1946	Manderville (Case 3)	Malignant melanoma	30	30	Pneumonectomy	No data			
1946	Manderville (Case 4)	Cherionoma, cervix	72	72	Lobectomy	No data			
1947	Aub and Mallory (Cable case 33,612) ²⁴	Hypernephroma	Not stated		Segmental resection	No data			
1947	Overholt and Langer ²⁵	Myxosarcoma, site?	Not stated		Lobectomy	No data			
1947	Major and Taylor ²⁶	Choriocarcinoma	30	48	No data	No data			
1947	Rienhoff ²⁷ (Case 1)	Fibrosarcoma, tendon sheath	Not stated		No data	No data			
1947	Rienhoff (Case 2)	Fibrosarcoma, tendon sheath	Not stated		Bilateral lobectomy	No	74	7	
1947	Alexander and Haight (Case 1)	Neurogenic sarcoma, ulnar nerve	18	23% to 13 (1)	Lobectomy	No	48		
1947	Alexander and Haight (Case 2)	Myogenic sarcoma, thigh	19	21	Double lobectomy	Yes	13		
1947	Alexander and Haight (Case 3)	Fibrosarcoma, thigh	24	20	Pneumonectomy	Yes			
1947	Alexander and Haight (Case 4)	Carcinoma, rectum	48	72	Pneumonectomy	No			
1947	Alexander and Haight (Case 5)	Spindle-cell sarcoma, leg	4	5	Pneumonectomy	Yes	1	13%	Impending (m)
1947	Alexander and Haight (Case 6)	Renal cell carcinoma	61	89%	Pneumonectomy	Yes			
1947	Sweet ²⁸ (Case 1)	Sarcoma, breast	48	48	Pneumonectomy	No data			
1947	Sweet (Case 2)	Carcinoma, ovary	120	120	Lobectomy	No data	84	12	12 plus
1947	Sweet (Case 3)	Fibrosarcoma, chest wall	26	26	Lobectomy	No data	0		
1947	Sweet (Case 4)	Renal cell carcinoma	3%	3%	Lobectomy	No data	15	1½	Unknown
1947	Sweet (Case 5)	Renal cell carcinoma	2	2	Lobectomy	No data		9	3 days
1947	Sweet (Case 6)	Renal cell carcinoma	24	24	Partial lobectomy	No data			
1947	Dolley ²⁹	Carcinoma, mandible	108	112	Lobectomy	No data		5 plus	10
1947	Carriso	Osteogenic sarcoma, thigh	40	58	Pneumonectomy	No data			
1947	Ransom ²¹	Carcinoma, colon	72	73½	Lobectomy	No	3½		
1948	Effer and Blades ³¹ (Case 1)	Malignant synovial, calf	24	2	Lobectomy	No data	17		
1948	Effer and Blades (Case 2)	Carcinoma, sigmoid	14	22	Lobectomy	No data	18 (n)	(n)	
1948	Effer and Blades (Case 3)	Teratocarcinoma, testis	10	17	Lobectomy	No data		4	

(Continued on following page.)

TABLE III. PULMONARY RESECTION FOR METASTASIS: MALIGNANT LESIONS: DATA ON SIXTY-TWO REPORTED CASES INCLUDING TEN MAYO CLINIC CASES

REPORT		PRIMARY LESION	TIME INTERVAL (MONTHS)		OPERATION	BRONCHIAL INVOLVEMENT	MONTHS AFTER PULMONARY RESECTION		UNTIL DEATH
YEAR	AUTHOR		PRIMARY TUMOR TO METASTASIS	REMOVAL OF PRIMARY TUMOR TO PULMONARY RESECTION			WITHOUT RECURRENCE	UNTIL RECURRENCE	
1927	Divis ³⁷	Sarcoma, breast	(u)	(a)	Remove metastasis	No data	6	(b)	
1930	Torek ⁸	Adeno-acanthoma, uterus	24	36	Remove metastasis	No data	No data		4 days
1933	Lilienthal ¹⁰	Sarcoma, uterus	(c)	(c)	Pneumonectomy	No		(d)	4 1/2
1937	Röpke ¹⁰ (Case 1)	Carcinoma, breast	36	36	Lobectomy	No data			
1937	Röpke (Case 2)	Carcinoma, ovary	29	36	Lobectomy	No data	42		
1939	Barney and Churchill ¹¹ (Case 1)	Renal adenocarcinoma	(e)	15	Partial lobectomy	No data	144		Well
1940	Churchill ¹³ (Case 2)	Not stated			Partial lobectomy	No data	No data	No data	
1940	Churchill (Case 3)	Not stated			Bilateral partial lobectomy (2 operations)	No data	No data	No data	
1940	Churchill (Case 4)	Not stated			Lobectomy	No data	No data	No data	
1940	Churchill (Case 5)	Not stated			Lobectomy	No data	No data	No data	
1940	Churchill (Case 6)	Not stated			Pneumonectomy	No data	No data	No data	
1941	Cotton ¹⁴	Adenocarcinoma, cecum	48	48	Pneumonectomy	Yes	36	No data	
1941	Raines ¹⁵	Carcinoma, sigmoid	9	21	Pneumonectomy	Yes	6	3	
1942	Freedlander and Greenfield ¹⁶ (Case 1)	Granulosa-cell tumor, ovary	(f)	(g)	Pneumonectomy	Yes			
1942	Freedlander and Greenfield (Case 2)	Adenocarcinoma, thyroid	480	480	Pneumonectomy	Yes			1 day
1943	King and Castleman ¹⁷	Carcinoma, stomach	(c)	(c)	Lobectomy	Yes		17	2 days
1943	Brezina and Lindskog ¹⁸	Carcinoma, uterus	156	162	Pneumonectomy	Yes			24
1944	Deming and Lindskog ¹⁹	Carcinoma, bladder and urethra	144 to 9	150 to 15	Pneumonectomy	No		2	5 1/2
1944	Blalock ²⁰ (Case 1)	Carcinoma, rectum	(h)	(h)	Pneumonectomy	No data			
1945	Tinney and McDonald ²¹ (Mayo Clinic, Case 1)	Hypernephroma	43	54	Pneumonectomy	Yes	37	66	81
1945	Edwards ²² (Case 1)	Squamous-cell epithelioma, palato	24	37	Pneumonectomy	Yes			
1946	Edwards (Case 2)	Carcinoma, rectum	Not stated	Not stated	Lobectomy	No data	No data	No data	15
1946	Edwards (Case 3)	Carcinoma, rectum	Not stated	Not stated	Pneumonectomy Wedge resection	No data	No data	No data	13

of pain in the chest. In 3 other patients the only finding was a positive result on roentgenologic examination of the chest. Two patients complained of a recent acute respiratory infection and 2 of wheezing.

Time Interval Between Removal of the Primary Tumor and the Appearance of the Metastatic Pulmonary Lesion.—This interval of time varied from eleven months to twenty-two years. In 2 cases, the metastatic growth was removed first. In 3 instances there were multiple excisions of the primary tumor before the appearance of the metastatic lesion.

Bronchial Involvement.—Gross bronchial involvement was demonstrated in 7, or 70 per cent, of the 10 cases. In 4 of the 10 cases, positive tissue evidence of malignancy was obtained by means of bronchoscopic biopsy.

Survival Periods.—Of the 10 patients, 8 are now dead and 2 are living and well with no evidence of recurrence. The longest survival following pulmonary resection for a metastatic lesion in this group was six years and nine months (Table II).

ANALYSIS OF DATA ON ALL SIXTY-TWO REPORTED CASES

This group includes the 52 cases reported in the literature plus the 10 Mayo Clinic cases reported herein. The pertinent data relative to these 62 cases have been arranged in tabular form and are listed by year of publication and author (Table III).

Type of Primary Tumor.—In 57 instances the type of the primary tumor was mentioned by the author. Of these, 18 were sarcomas, 35 were carcinomas, and 4 were of the mixed tumor type. Of the 4 mixed tumors, 1 was a mixed tumor of the jaw, 1 was a mixed squamous-cell epithelioma and adenocarcinoma of the testis, and 2 were teratoma of the testis. Inasmuch as the carcinomatous element predominated in all of these tumors, they have been classified among the carcinomas for the sake of simplicity (Table IV). A review of this information indicates that the most common primary site in the entire series was carcinoma of the large bowel (11 cases); of these, 6 were rectal carcinomas. The primary tumor was hypernephroma in 7 instances and fibrosarcoma in 7 instances. Carcinoma of the ovary was fourth in frequency, with the lesion in 5 cases being primary in this organ.

Time Interval Between Removal of the Primary Tumor and the Appearance of the Metastatic Pulmonary Lesion.—This, of course, was extremely variable ranging from zero to forty years (Freedlander and Greenfield's Case 2).¹⁰ In several instances the metastatic lesion was discovered prior to the primary growth or simultaneously. Although the primary carcinoma of the thyroid was removed forty years before in the case referred to previously, a small local recurrence of the tumor in the thyroid gland was found at necropsy. In Diviš's case, the patient had undergone ten local removals of a sarcoma of the breast over a period of thirty-five years before the occurrence of the pulmonary lesion. Deming and Lindskog's¹¹ patient also had multiple removals of the primary neoplasm of the bladder and urethra over a twelve-year period. Thus, the true time interval before pulmonary metastasis occurred cannot be accurately estimated in these cases. In 7 instances, the interval of time elapsing

REPORT		PRIMARY LESION	TIME INTERVAL (MONTHS)		REMOVAL OF PRIMARY TUMOR TO PULMONARY RESECTION	OPERATION	BRONCHIAL INVOLVE- MENT	MONTHS AFTER PULMONARY RESECTION		UNTIL RE- CURRENCE	UNTIL DEATH
YEAR	AUTHOR		PRIMARY TUMOR TO METASTASIS	PRIMARY TUMOR TO PULMONARY RESECTION				WITHOUT RE- CURRENCE	WITH RE- CURRENCE		
1948	Blalock ³¹ (Case 2)	Adenocarcinoma, cecum	39	48	Pneumonectomy	No data	5				
1948	Flick ³²	Liposarcoma, thigh	96	96	Lobectomy	No data	16	20	24		
1948	Mayo Clinic (Case 2)	Adenocarcinoma, rec- tum	(f)	(g)	Pneumonectomy	Yes					
1948	Mayo Clinic (Case 3)	Adenocarcinoma, jejunum	(e)	(g)	Pneumonectomy	No				23 days (p)	
1948	Mayo Clinic (Case 4)	Teratoma, testis	11	15	Lobectomy	No		18	21		
1948	Mayo Clinic (Case 5)	Adenocarcinoma, rec- tum	50	51	Lobectomy	Yes		8	12		
1948	Mayo Clinic (Case 6)	Osteogenic sarcoma, os calcis	(q)	(q)	Lobectomy	Yes		3(q)	3 plus		
1948	Mayo Clinic (Case 7)	Adenocarcinoma of jaw, mixed tumor type	(r)	270 to 234	Pneumonectomy	Yes		5	12		
1948	Mayo Clinic (Case 8)	Mixed squamous-cell and adenocar- cinoma, testis	36	36	Pneumonectomy	Yes		2	4		
1948	Mayo Clinic (Case 9)	Fibrosarcoma, leg	(s)	132 to 84 (s)	Lobectomy	No	28		Well		
1948	Mayo Clinic (Case 10)	Adenocarcinoma, ovary	42	48	Lobectomy	Yes	24		Well		

- (a) Ten operations over a 34-year period (from the age of 7 to 41 years) for removal of recurring sarcoma of breast. First removal, 35 years previously; last removal, 1 year previously.
- (b) Followed up 6 months.
- (c) Metastatic lesion removed first; necropsy revealed primary growth.
- (d) Cerebral metastasis occurred after several months.
- (e) Metastatic lesion discovered first.
- (f) Primary and secondary lesions discovered simultaneously.
- (g) Metastatic tumor removed first.
- (h) Multiple operations for recurring carcinoma of bladder and urethra over a 12-year period.
- (i) Untraced after 6 months.
- (j) During the 5-year period prior to pneumonectomy, 4 local excisions of the primary tumor had been done, culminating in amputation 1 year prior to pulmonary resection.
- (k) Too recent to evaluate. Patient still living but hilar nodes involved.
- (l) Two operations on metastatic lesions 13 months apart.
- (m) Interval not sufficient to permit evaluation; prognosis seems poor.
- (n) An intracutaneous metastatic lesion was removed by craniotomy 6 months after pulmonary resection; patient well 1½ years after death due to rupture of previously silent carcinoma of the jejunum, the primary lesion, found at necropsy.
- (o) First operation on primary lesion 23 years before; second operation on primary lesion 4 years previously; lobectomy was frankly palliative.
- (p) First operation on primary lesion 23 years before; second operation on primary lesion 4 years previously; third operation for removal of primary growth performed at time of pulmonary resection.

or branch by the metastatic tumor. In 17 cases (63 per cent), bronchial involvement was present and in 10 cases it was not. By inference it may be assumed that in most of the remaining cases in which this feature was not discussed, gross bronchial involvement was probably not present. For the entire series of 62 cases, the incidence of bronchial involvement as definitely stated was 27 per cent (17 out of 62 cases). The true percentage undoubtedly lies somewhere between this figure and the previously mentioned 63 per cent, probably 35 to 40 per cent of cases.

Of the 17 cases in which bronchial involvement was noted in this series, there was a positive result on bronchoscopic biopsy in 10 (59 per cent). This is an incidence of 16 per cent for positive results on biopsy for the entire series.

Type of Operation Performed.—Of the 62 patients reported on, 27 had lobectomy, 25 had pneumonectomy, and 10 had partial lobectomy or segmental resection. Two patients had multiple removal of pulmonary metastatic tumors, of which 1 was by lobectomy on two separate occasions, and 1 by bilateral partial lobectomy. Two instances listed as segmental resection were probably simple excision of the metastatic nodule. In the cases reported early, partial lobectomy and local excision were not infrequently performed, whereas pneumonectomy was usually the procedure of choice in the cases in which operation was performed during the middle years. More recently, lobectomy has been done more often than any other procedure, and superficially it appears that the best results have been obtained from this procedure (Table VI). It is impossible to convey accurately the entire picture by this means, however, inasmuch as the figures do not take into consideration such important factors as extent of the lesion at the time of operation, operative and postoperative mortality, and the time interval between discovery of the metastatic growth and surgical excision. It is probably true, nevertheless, that in the case of a solitary metastatic tumor well within the confines of one lobe, lobectomy is the procedure of choice. Segmental or wedge resection may occasionally be considered in selected cases.

TABLE VI. SURVIVAL ACCORDING TO TYPE OF OPERATIVE PROCEDURE CARRIED OUT

OPERATIVE PROCEDURE	PATIENTS		
	OPERATED ON	SURVIVING	
		NUMBER	PER CENT
Partial or segmental lobectomy	10	3	30
Lobectomy	27	13	48
Pneumonectomy	25	7	28

End Results.—Of the 62 patients who underwent pulmonary resection for metastatic lesions, 23 were living and well at the time of the various reports (37 per cent). This figure is obviously too high inasmuch as some of the patients had been followed for only a short time. Furthermore, some of the patients who were well at the time of reporting will likely have died subsequently of further spread of their disease. In an occasional instance, the patient was alive and well but the author commented upon the unfavorable prognosis in view of involved hilar glands at the time of operation, or other

TABLE IV. TYPES OF PRIMARY TUMORS FOR WHICH PULMONARY RESECTION OF THE METASTATIC LESION WAS CARRIED OUT (ALL REPORTED CASES)

TYPE OF PRIMARY TUMOR	CASES
Carcinoma (68 per cent)	39
Adenocarcinoma of large bowel (cecum 2, sigmoid 2, colon 1, rectum 6)	11
Hypernephroma	7
Carcinoma of ovary (adenocarcinoma 3, granulosa-cell carcinoma 2)	5
Carcinoma of uterus (adenocarcinoma 1, adenocarcinoma 1)	2
Mixed tumor type (teratoma of testis 2, squamous-cell epithelioma and adenocarcinoma of testis 1, mixed tumor of jaw 1)	4
Carcinoma of cervix	1
Chorionepithelioma	1
Carcinoma of breast	1
Carcinoma of thyroid	1
Carcinoma of stomach	1
Adenocarcinoma of jejunum	1
Carcinoma of bladder and urethra	1
Squamous-cell epithelioma of palate	1
Carcinoma of mandible (adamantinoma)	1
Malignant melanoma	1
Sarcoma (32 per cent)	18
Fibrosarcoma (tendon sheath 2, leg 2, thigh 1, arm 1, chest wall 1)	7
Osteogenic sarcoma (thigh 1, os calcis 1)	2
"Sarcoma" (breast 2, uterus 1, fibula 1)	4
Myxosarcoma	1
Neurogenic sarcoma	1
Myogenic sarcoma	1
Liposarcoma	1
Malignant synovioma of calf	1

between removal of the primary tumor and the appearance of the metastatic lesion in the lung was, or could have been, ten years or longer (Table V). The usual time interval was much less than the extremes indicated in Table V, however, averaging one to four years. In some instances the metastatic growth was evident in a matter of only a few months. In the group of 7 "long interval" cases, 5 patients died, 1 was alive two years after pulmonary resection, and the remaining 1 was followed only six months.

Bronchial Involvement.—In 27 of the 62 cases, a definite statement was given concerning the presence or absence of involvement of a major bronchus

TABLE V. TIME INTERVAL BETWEEN REMOVAL OF PRIMARY TUMOR AND APPEARANCE OF PULMONARY METASTATIC LESION IN SEVEN CASES IN WHICH DURATION WAS LONGEST

AUTHOR	INTERVAL (YEARS)
Freedlander and Greenfield ¹⁰ , Case 2	0 to 40
Divi ⁸ *	1 to 35
Mayo Clinic, Case 7*	19 to 22
Brezina and Lindskog ¹⁸	13
Deming and Lindskog ¹⁹	1 to 12
Mayo Clinic, Case 9*	7 to 11
Sweet, ² Case 2	10

*Multiple operations performed for recurring primary tumor.

TABLE VIII. PRIMARY LESION IN TWENTY-THREE CASES IN WHICH THE PATIENT WAS ALIVE AT THE TIME THE CASE WAS REPORTED

TYPE OF TUMOR	CASES
Carcinoma	13
Large bowel (cecum 2, sigmoid 1, colon 1, rectum 1)	5
Hypernephroma	3
Ovary	3
Cervix	1
Chorionepithelioma	1
Sarcoma	10
Fibrosarcoma (thigh, leg, chest wall)	3
Breast	2
Osteogenic, fibula	1
Neurogenic, ulnar nerve	1
Myogenic, thigh	1
Liposarcoma, thigh	1
Malignant synovium	1

In 10 instances in the group of 23 surviving patients, the presence or absence of bronchial involvement was commented upon by the author. Of this number, 5 patients showed invasion of a major bronchus by the tumor and 5 did not.

In some instances in which there was a final fatal outcome, pulmonary resection resulted in such a degree of palliation and prolongation of life as to make the procedure definitely worth while. In the Mayo Clinic Case 1, for example, the patient lived six years and nine months before eventually dying of further metastasis, and most of this time she was well. Usually the interval between pulmonary resection for a metastatic lesion and death has been of short duration. Thus, it appears that in the majority of cases, if further spread is going to occur after pulmonary resection, it will probably be apparent within a period of several months to one and one-half years or two years.

Six operative or immediate postoperative deaths occurred in the entire series of 62 cases, an incidence of 10 per cent.

SUMMARY

Reports of 52 cases in the literature plus the records of 10 cases at the Mayo Clinic in which pulmonary resection was performed for metastatic malignant lesions have been studied and the findings presented.

The primary tumor was sarcoma in 32 per cent (18 cases), and carcinoma in 68 per cent (39 cases) of the 57 cases in which the type of primary tumor was mentioned. The primary tumors for which resection of solitary pulmonary metastatic lesions was most frequently done were carcinoma of the large bowel (11 cases), hypernephroma (7 cases), fibrosarcoma (7 cases), and carcinoma of the ovary (5 cases).

The time intervals between removal of the primary tumor and appearance of the secondary lesion in the lung were extremely variable, ranging from zero to forty years. With several exceptions, the patients with long intervals of time elapsing between the occurrence of the primary and the secondary tumors did no better than the group as a whole.

TABLE VII. DATA ON TWENTY-THREE PATIENTS WHO UNDERWENT PULMONARY RESECTION FOR A METASTATIC MALIGNANT LESION OF THE LUNG AND WHO WERE ALIVE WHEN THE CASES WERE REPORTED

AUTHOR	SURVIVAL (MONTHS)	PRIMARY TUMOR
Diviš ⁷	6	Sarcoma of breast
Röpke, ¹⁰ Case 2	42	Carcinoma of ovary
Barney and Churchill ¹¹	144	Hypernephroma
Cotton ¹⁴	36	Carcinoma of cecum
Freedlander and Greenfield, ¹⁶ Case 1	6	Granulosa-cell carcinoma of ovary
Blalock, ²⁰ Case 1	37	Carcinoma of rectum
Edwards, ²² Case 4	216	Sarcoma of fibula
Mandeville, ²³ Case 4	3 (?)	Carcinoma of cervix
Maier and Taylor ²⁶	36	Chorionepithelioma
Alexander and Haight, ¹ Case 1	74	Neurogenic sarcoma of ulnar nerve
Alexander and Haight, Case 2	48	Myogenic sarcoma of thigh
Alexander and Haight, Case 3	13	Fibrosarcoma of thigh
Alexander and Haight, Case 6	1	Hypernephroma
Sweet, ²⁸ Case 1	84	Sarcoma of breast
Sweet, Case 3	9	Fibrosarcoma of chest wall
Sweet, Case 4	15	Hypernephroma
Samson ³¹	3½	Carcinoma of colon
Effler and Blades, ² Case 1	17	Malignant synovium
Effler and Blades, Case 2	18	Carcinoma of sigmoid
Blalock, ³⁴ Case 2	5	Carcinoma of cecum
Flick ³²	16	Liposarcoma of thigh
Mayo Clinic, Case 9	28	Fibrosarcoma of leg
Mayo Clinic, Case 10	24	Adenocarcinoma of ovary

modifying circumstances. In 1 instance the patient subsequently had an intracranial metastatic lesion which was removed by craniotomy (Effler and Blades,² Case 2). The patient was alive and well one and one-half years after lobectomy, and thirteen months after craniotomy. This is indeed carrying extirpative surgery to great lengths.

Study of the surviving 23 patients yields certain interesting information (Table VII). It appears, for instance, that Edwards'²² patient (Case 4) has survived the longest to date, namely, eighteen years. Unfortunately, this case was not described in any detail. The next longest survival was the patient of Barney and Churchill¹¹ who was alive and well twelve years later. The operation in the latter case was done for metastatic hypernephroma, and in Edwards' case it was done for sarcoma of the fibula. Sweet²⁸ has had a seven-year survival following resection for metastatic sarcoma of the breast, and Alexander and Haight's¹ first patient was alive and well six years and two months subsequent to resection for a metastatic neurogenic sarcoma. It is noteworthy that 3 of these 4 long survivals represented cases of sarcoma and the fourth represented a hypernephroma, a tumor well known to give rise to solitary metastatic lesions of the lung. Partial lobectomy was the operative procedure carried out in Edwards'²² case and in Barney and Churchill's¹¹ case, while pneumonectomy was done in Sweet's²⁸ case, and bilateral lobectomy was performed in the case of Alexander and Haight.¹ The last-mentioned patient had 2 metastatic growths removed thirteen months apart and was alive and well six years and two months later.

Table VIII gives the distribution of the primary lesion according to site in the 23 cases in which the patient was alive at the time of the report.

2. Effler, D. B., and Blades, Brian: Surgical Treatment of the Solitary Lung Metastasis, *J. THORACIC SURG.* 17: 27-37, 1948.
3. Seiler, H. H.: Pulmonary Resection for Metastatic Malignancy, Thesis, Graduate School of the University of Minnesota, 1948, 142 pp.
4. Gerulanos, M.: Eine Studie über den operativen Pneumothorax in Anschluss an einen Fall von Lungenresection wegen Brustwandarkom, *Deutsche Ztschr. f. Chir.* 49: 497-536, 1898.
5. Sedillot: Quoted by Gerulanos.¹
6. Meyer, Willy: Observations on Lung Suppuration and Its Treatment; a Brief Review of Personal Experiences and Clinical Impressions Obtained Within the Last Twelve Years, *Arch. Surg.* 6: 361-425, 1923.
7. Divis, Georg: Ein Beitrag zur operativen Behandlung der Lungengeschwülste, *Acta chir. Scandinav.* 62: 329-341, 1927.
8. Torek, Franz: Removal of Metastatic Carcinoma of the Lung and Mediastinum; Suggestions as to Technic, *Arch. Surg.* 21: 1416-1424, 1930.
9. Lillenthal, Howard: Pneumectomy for Sarcoma of the Lung in a Tuberculous Patient, *J. THORACIC SURG.* 2: 600-615, 1933.
10. Röpke, W.: Mehrjährige Heilung nach Operation eines Lungenkarzinoms, *Zentralbl. f. Chir.* 64: 803-806, 1937.
11. Barney, J. D., and Churchill, E. D.: Adenocarcinoma of the Kidney With Metastasis to the Lung Cured by Nephrectomy and Lobectomy, *J. Urol.* 42: 269-276, 1939.
12. Barney, J. J. D.: Twelve-Year Cure Following Nephrectomy for Adenocarcinoma and Lobectomy for Solitary Metastasis, *J. Urol.* 52: 406-407, 1944.
13. Churchill, E. D.: Resection of the Lung, *Surgery* 8: 961-991, 1940.
14. Cotton, B. H.: Differential Diagnosis of Primary and Metastatic Malignancy of the Lung, *Am. J. Surg.* 54: 173-178, 1941.
15. Raine, Forrester: Metastatic Carcinoma of the Lung Invading and Obstructing a Bronchus, *J. THORACIC SURG.* 11: 216-218, 1941.
16. Freedlander, S. O., and Greenfield, Jack: Hemoptysis in Metastatic Tumors of the Lung Simulating Bronchiogenic Carcinoma; a Report of Two Cases, *J. THORACIC SURG.* 12: 100-116, 1942.
17. King, D. S., and Castleman, Benjamin: Bronchial Involvement in Metastatic Pulmonary Malignancy, *J. THORACIC SURG.* 12: 305-315, 1943.
18. Brezina, P. S., and Lindskog, G. E.: Total Pneumectomy for Metastatic Uterine Carcinoma, *J. THORACIC SURG.* 12: 728-733, 1943.
19. Deming, C. L., and Lindskog, G. E.: Papillomatosis of Bladder and Entire Urethra; Infiltrating Cancer of Bladder. Late Pulmonary Metastasis. Successful Pneumectomy, *Tr. Am. A. Genito-Urin. Surgeons* 37: 30-50, 1944.
20. Blalock, Alfred: Recent Advances in Surgery, *New England J. Med.* 231: 261-267; 293-300, 1944.
21. Tinney, W. S., and McDonald, J. R.: Pulmonary Metastasis of Carcinoma Diagnosed by Bronchoscopy, *Minnesota Med.* 28: 554-558, 1945.
22. Edwards, A. T.: Carcinoma of the Bronchus, *Thorax* 1: 1-25, 1946.
23. Mandeville, F. B.: Roentgen and Clinical Problems in So-called Solitary Metastatic Tumors in the Chest, *Am. J. Surg.* 71: 669-675, 1946.
24. Massachusetts General Hospital: Case 33012, *New England J. Med.* 236: 38-40, 1947.
25. Overholt, R. H., and Langer, Lazaro: A New Technique for Pulmonary Segmental Resection, *Surg., Gynec. & Obst.* 84: 257-268, 1947.
26. Maier, H. C., and Taylor, H. C., Jr.: Metastatic Chorionepithelioma of Lung Treated by Lobectomy, *Am. J. Obst. & Gynec.* 53: 674-677, 1947.
27. Blenkhoff, W. F., Jr.: The Present Status of the Surgical Treatment of Carcinoma of the Lung, *Ann. Surg.* 125: 541-565, 1947.
28. Sweet, R. H.: Quoted by Alexander and Haight.¹
29. Dolley, F. S.: Quoted by Alexander and Haight.¹
30. Carr, Duane: Quoted by Alexander and Haight.¹
31. Samson, P. C.: Quoted by Alexander and Haight.¹
32. Flick, J. B.: Quoted by Effler and Blades.²
33. Willis, R. A.: The Spread of Tumors in the Human Body, London, 1934, J. & A. Churchill, Ltd., 540 pp.
34. Blalock, Alfred: Quoted by Effler and Blades.²

DISCUSSION

DR. D. H. WATERMAN, Knoxville, Tenn.—Dr. Seiler has shown that isolated metastatic lesions in the lung are not uncommon. In bronchiogenic carcinoma, however, metastasis limited to the contralateral lung is relatively rare. I rise to report an instance in this latter

Bronchial involvement was noted in 63 per cent of the cases in which the reports especially mentioned this feature, or in 27 per cent of the entire group. The true percentage probably lies somewhere between these two figures, indicating that in a very substantial proportion of cases the tumor will be found to be invading a major bronchus. This is in contradistinction to the former teaching that bronchial invasion by a metastatic tumor is uncommon. A positive diagnosis was made by means of bronchoscopic biopsy in 16 per cent of the entire series.

The type of operation performed apparently did not too greatly influence the survival rate. Lobectomy or segmental resection is probably the procedure of choice when possible.

Twenty-three patients out of 62 were living and well at the time reported on (37 per cent). Two of these 23 had survived ten years or longer, and 7 more had survived for periods ranging from three to ten years. The remaining 14 patients had been living and well for periods varying from several months to two years after resection. Of the group of 23 survivors, the primary lesion was sarcoma in 10 instances and carcinoma in 13. The most common primary tumors encountered among the surviving group were carcinoma of the colon and rectum, hypernephroma, fibrosarcoma, and carcinoma of the ovary.

Palliative pulmonary resection for metastatic malignant lesions occasionally seemed to be justifiable on the basis of prolongation of life (six years and nine months in one instance) and the degree of comfort afforded the patient.

The operative mortality rate for the series was 10 per cent.

CONCLUSION

The surgical excision of solitary, metastatic, malignant lesions of the lung is indicated in certain cases in which the primary tumor apparently has been completely removed and in which there is no evidence of further metastatic spread. Experience has shown that in a few instances survival for many years may be expected to follow such a procedure, while at other times the degree of palliation afforded makes the attempt justifiable.

ADDENDUM

Since the completion of this work, pulmonary resection for metastatic lesions has been performed in 6 more cases at the Mayo Clinic. The primary lesions in this group included 2 fibrosarcomas (chest wall), 1 osteogenic sarcoma, 1 hypernephroma, 1 adenocarcinoma of the uterus, and 1 adenocarcinoma of the maxillary antrum (cylindroma type). Pneumonectomy was carried out in 2 instances and lobectomy in four. Bronchial involvement was noted in 2 cases. Two patients have since died of recurrence of their malignant disease at eight months and one year, respectively. The remaining 4 patients are alive and have had no evidence of return of their disease for periods of eighteen months, eight months, eight months, and four months, respectively.

REFERENCES

1. Alexander, John, and Haight, Cameron: Pulmonary Resection for Solitary Metastatic Sarcomas and Carcinomas, *Univ. Hosp. Bull., Ann Arbor* 12: 117-118, 1946; *Surg., Gynec. & Obst.* 85: 129-146, 1947.

of thoroughness with which the body is explored for possible demonstrable metastases other than the obvious one in the lung, whether there are other metastases which are not demonstrable and which, if present, presumably cannot be removed.

Dr. Haight and I felt that it would be of interest if a follow-up were presented to you concerning the patients we reported upon in 1947 (slide). There are not grouped chronologically but according to the type of lesion and, incidentally, a few have been added since 1947. The largest group by far, in contradistinction to Dr. Seiler's patients, had sarcomas. The first group is of sarcoma, the second of carcinoma, and the last, a single case, is a cystoma of the ovary which microscopically was considered to be benign but which had metastasized to the lung. Two of the living patients have new metastases which are irremovable and, of course, these patients will die. Among the whole group of 11 patients, 4 are dead, 2 will almost certainly die, and 5 are in excellent health with no sign or symptom of malignancy.

Our first and most interesting patient, and the one who convinced us that the gamble is worth while, is a woman who had a very malignant type of spindle-cell sarcoma of the median nerve, which was removed by Dr. Alexander T. Bunts at the Cleveland Clinic nine and one-half years ago. One year later a small lesion appeared in the right lower lobe; Dr. Bunts explored the forearm because of causalgia and found a benign fibroma without any evidence of recurrence. I removed the right lower pulmonary lobe and one year later the patient returned for a check-up examination because of cough; she had a large lesion in the left upper lobe. Ill luck had returned; more than one metastatic lesion had been present and, possibly, a great many. I decided, however, to remove the left upper lobe on the remote chance that it contained the only remaining malignant tissue; the patient is now perfectly well eight and one-half years after the second lobectomy.

The second patient is also of special interest; he had had two operations on his thigh for a myogenic sarcoma, the second of which was probably for an implantation lesion; before I removed the right upper lobe, I had biopsy specimens taken of the thigh—three deep slices to the femur from which 50 sections were prepared and no sarcoma cells were found. The reason for a strong suspicion of recurrence locally at the site of the primary lesion was that this patient had inadvisably been given very heavy doses of x-ray therapy following removal of the thigh sarcoma, and there was deep ulceration so that it was impossible to tell from inspection and palpation whether there had been a recurrence. But there was none and he is well and working, six and three-quarter years after operation, and without any sign of another metastatic lesion.

We have learned very little since our first patient of nine and one-half years ago as to what criterion, other than the obvious ones that occur to us all, are important with regard to a reasonable expectation of a cure or possibly of prolonged relief of distressing symptoms.

DR. G. N. J. SOMMER, JR., Trenton, N. J.—It is interesting to note that Weinlechner* in 1881 removed two metastatic tumors from the lung at the time of resection of a huge sarcoma of the bony thoracic wall. The patient died postoperatively. Krönlein in 1883,† removed a metastatic sarcoma in the lower lobe of the lung when operating for a recurrent primary tumor of a rib. The patient lived for six years and died of a further recurrence of the costal sarcoma without further apparent pulmonary lesions. In both these cases the metastatic tumors were definitely hematogenous and did not involve the lungs by continuity.

The pathologic basis on which metastatic tumors may be successfully treated has been laid by Schmidt and Muir. Schmidt‡ showed that in the presence of visceral carcinoma many tumor cells pass to the lungs. Most of these tumor emboli, however, do not survive or, if they do grow, remain of microscopic size and cannot be disclosed without very careful examination of the lungs. Furthermore, peripheral metastases arise, not from tumor emboli passing through the pulmonary capillaries, but from the pulmonary metastatic tumors, which may be the microscopic ones described. It is also from such microscopic pulmonary metastatic tumors that

*Wien. med. Wochschr. 22: 589, 1882.

†Berliner. klin. Wochschr. 21: 129, 1884.

‡Die Verbreitungswege der Karzinome, Jena, 1903.

group, in which we were confronted with the problem of an apparent metastasis arising in the opposite lung of a patient on whom a pneumonectomy had been performed some five and one-half years previously (slides).

The patient is a 53-year-old white man whom we first saw in October, 1942, with elevation of the left diaphragm and a six months' history of weight loss, cough, and other typical symptoms of bronchiogenic malignancy. Bronchoscopy at that time revealed a squamous-cell carcinoma blocking the left lower lobe bronchus. Following removal of biopsy tissue, the left lower lobe aerated and the diaphragm descended to its normal position. In November, 1942, total pneumonectomy was performed on the left using individual ligation technique. There was no evidence of mediastinal metastases at operation. Dyspnea did not develop subsequently, so thoracoplasty was not performed. The patient was followed at three-month intervals, and apparently did well, showing no signs of recurrence. However, in March, 1949, as shown in this slide, a small rounded lesion $1\frac{1}{4}$ by 2 cm. appeared in the right upper lobe. We thought there was a possibility of recurrent malignancy, but with perhaps wishful thinking, hoped it might be of some other origin. After a trial period of bed rest, however, the lesion was seen to be slightly larger. In view of this, we decided we were dealing with metastatic tumor, and suggested exploration. We were rather reluctant, of course, in such an instance to propose surgery, realizing the potential risks involved in any operative attack on a single lung. Unable to find a similar experience reported in the literature, we gave considerable thought as to what type of procedure would be advisable, first considering a preliminary operation to promote pleural adherence in the area. We rejected this because of the limitations it imposed, and reached the conclusion that open thoracotomy would be the procedure of choice.

The next slide showing the roentgenogram taken just before surgery indicates further enlargement of the lesion.

The patient was operated upon on Sept. 10, 1948, under intratracheal cyclopropane. A posterolateral incision was made and the pleural cavity entered through the fifth intercostal space without division of either the fifth or sixth ribs. This approach was used so there would be as little interference as possible with the function of the remaining hemithorax. No adhesions were present. The right lung was kept fully inflated with positive pressure throughout the operation to insure adequate aeration. The lesion was found to be a firm, round tumor mass, somewhat smaller than a walnut, located in the posterior aspect of the right upper lobe just above the fissure. A wedge segmental resection was performed between clamps, removing about 2.5 cm. of lung tissue on either side of the tumor.

A fenestrated catheter was inserted along the gutter to a point just above the area of resection, and continuous suction instituted postoperatively for two days. Fortunately the postoperative course was uneventful and recovery took place without incident.

These color slides show the gross specimen and the microscopic appearance. The pathologist reported the tumor to be apparently a metastatic squamous-cell bronchiogenic carcinoma.

This last slide is the present situation six months after operation, showing some residual fibrosis in the area of the resected lesion. The man has had no evidence of difficulty since operation, and has returned to active business life; he is an ardent fisherman and hunter, and has taken up these sports as well.

I think I might say in conclusion that we are perhaps open to criticism for procrastinating in this case before proceeding to surgery. I believe we are justified in saying that the appearance of a circumscribed lesion in the lung following previously known carcinoma should always be considered a metastatic lesion until proved otherwise.

DR. JOHN ALEXANDER, *Ann Arbor*.—The patients and the surgeons who deal with this condition are confessed gamblers. Before deciding whether or not to remove the pulmonary metastatic lesion, a biopsy of the site from which the primary lesion was removed (if this site is relatively superficial and accessible) will indicate whether the primary lesion has been completely removed. But it is pure guesswork, apart from the thoroughness or lack

A second boy who had a sarcoma of the leg, an osteogenic sarcoma, removed nine years before, developed pain and swelling in the remaining ankle, and when the roentgenologist took the x-ray picture and saw that he had but one leg he suspected pulmonary osteoarthropathy and took a picture of the chest. He found a very dense shadow in the upper left lobe. We removed the upper lobe in 1941 and the patient has had no recurrence of trouble and is entirely well.

We have had two other patients with sarcoma, one three years ago, one two years ago; both had sarcoma of the arm and had had recurrences and developed a lesion in the chest. Both have had lobectomies and are still well. We had another case on which I cannot tell you the final result; it was four years ago, and for two years he was well. The tumor was of enormous size and the original tumor was in the popliteal space. The last case is too recent; recurrence was present within a year after removal of an osteogenic sarcoma of the left leg. There is no local recurrence but she failed to appear at the dispensary for follow-up from last June until February, when this lesion was discovered. It had become of enormous size, but lobectomy with partial resection of the diaphragm was done.

I believe that probably sarcomas, because of their nature, being a low-grade disease as mentioned by the essayist, do offer more than the epithelial tumors.

DR. ADRIAN LAMBERT, New York.—I have one case that I think would be of interest. Three years ago I resected a 26-year-old girl's left upper lobe for a large globular adenocarcinoma that was four inches in diameter. We felt at the time that no lymph glands were involved; eight or ten were sent to the laboratory for section and were negative. We felt we were around the tumor until the paraffin sections came back and showed there were cells in one of the pulmonary veins. We therefore suspected that possibly she would develop metastases thereafter. Four months later she developed a lump on the thigh which I excised; it turned out to be just fibrous tissue. Eight months after this time, she developed a small, 1 cm. size nodule in the right lung. It was in the right upper lobe. Exploration for that was carried out one year after the original left upper lobe lobectomy and a right upper lobe lobectomy was performed. The tumor was exactly the same type of tissue as was seen in the original adenocarcinoma.

She has now gone two years and is living and well.

recurrences arise after resection of apparently solitary metastatic tumors of the lung. Muir* emphasized the importance of tissue resistance to metastatic tumors in limiting their growth and spread. Willist concurs in the views of Schmidt and Muir.

A case under the care of the speaker illustrates the views already outlined. The 52-year-old male patient had complained of thoracic pain for one year. Roentgenographic examination showed the lesion, which was thought to be of an inflammatory nature, involving the right seventh rib. It was explored with local anesthesia. Histologic examination disclosed an adenocarcinoma. Because of vague abdominal symptoms and the demonstration of several colonic diverticula, it was thought that the primary tumor was probably a minute one in the gastrointestinal tract. Roentgen ray therapy was administered to the costal tumor. After discussion of a similar case with Dr. J. R. Trimble of Baltimore, however, the seventh and the adjacent two ribs were widely resected. The patient lived for two years. Further studies failed to disclose a primary tumor. He died suddenly of gastrointestinal hemorrhage. The autopsy was performed in a mortician's establishment. The primary adrenal tumor was found above the right kidney. The fatal hemorrhage had come from an ulcer of the pyloric end of the stomach. Material for microscopic sections of the lung was taken from a caseous apical focus and foci considered to be pneumonic at the base of the right lung. The histologic sections of the second portion of lung, however, showed adenocarcinoma similar to that of the adrenal and rib. Numerous roentgenographic studies of the lungs had never shown evidence of metastatic tumor. No other gross secondary tumors were found in the post-mortem examination. Effler and Blades† have described a case in which a solitary pulmonary metastatic tumor was removed and later an apparently solitary one of the brain.

DR. ARTHUR AUFSES, New York.—I think there is one subject that has not been touched upon; that is the possibility that the solitary lesion that is seen in the lung after the patient has been known to have a carcinoma, may not be metastasis; it may be another primary tumor. That should be kept in mind in thinking about exploration for removal of a single lesion in the lung under such circumstances. We have recently had two patients one of whom had been treated for a number of years for a squamous-cell carcinoma of the lip, and who developed a solitary nodule in the lung. On removal of this lobe—we did a lobectomy because we felt that it was a metastasis—it was found to be a primary lesion of different nature from that of the lip, an adenocarcinoma. Another patient had had a carcinoma of the colon removed eight years previously, then a gastrectomy for carcinoma of the stomach two years previously, and then came in with a solitary lesion of the lung. A pneumonectomy was performed and the lesion in the lung was squamous-cell carcinoma, whereas the other two were adenocarcinomas. For that reason we feel that any solitary lesion must be considered to be possibly another primary carcinoma.

DR. WILLARD VAN HAZEL, Chicago.—I believe that the mesothelial metastatic lesions probably promise us more than the epithelial ones. We have had four patients with epithelial metastases, and only one is living, but she has other metastases. On the other hand we have six cases of sarcoma; the first was one that I reported not as a metastatic lesion but as a primary lesion of pulmonary osteoarthropathy in 1939. This girl had a grapefruit size mass in the chest which we removed; there was only a stalk about the size of a pencil which was fused with the fissures on the right side. This was reported as a fibroma, but the pulmonary osteoarthropathy returned in 1940. Little could be seen on the roentgenogram and because of the diagnosis of fibroma we did not suspect a recurrence, but there was nodulation at the hilus. On exploration we found nodes at the hilus and we did a resection, removing the lung in 1941. Since that time, three years ago, this girl has developed a lesion over the deltoid and we removed a similar tumor, undoubtedly metastatic, so the pneumonectomy did not cure her. Two years ago she had a popliteal tumor of similar character and it could be removed. She is still living and able to do things, although not in the best of health.

*Textbook of Pathology, ed. 4, Baltimore, 1936. William Wood & Company.

†Pathology of Tumors, St. Louis, 1948, The C. V. Mosby Company.

‡J. THORACIC SURG. 17: 27, 1948.

TABLE I. INCIDENCE OF SYMPTOMS PREVIOUS TO DIAGNOSIS

SYMPTOMS	MONTHS				TOTAL WITH SYMPTOM
	1 TO 6	6 TO 12	12 TO 18	18 PLUS	
Hemoptysis	11	6	5	3	25
Cough and sputum	9	12	6	5	32
Pleuritic pain	2	8	0	0	10
Emaciation	9	8	6	3	26
Fever and night sweats	6	4	3	1	14
No symptoms	-	-	-	-	4
No physical findings	-	-	-	-	7

had fever and night sweats of varying degree only 1 of the cases was found to have tuberculosis at autopsy. As would be expected the greater number had specific symptoms recognizable within the one-year period previous to diagnosis. Although the peripherally located group represents only approximately 20 to 25 per cent of all bronchiogenic carcinomas, 18, or 45 per cent, of the resected tumors of this series were in this group. This is explained by the fact that some of these tumors were found on routine fluoroscopic examination before symptoms had developed. Ten of this group had pleuritic pain. The unreliability of these symptoms and physical findings is well demonstrated in the preceding table and discussion. This same experience has been observed by many other surgeons.^{2, 3, 4}

COMMENTS ON TABLE II

TABLE II. LABORATORY OR OBJECTIVE EVIDENCE OF TUMOR

ROENTGENOGRAM	NUMBER	PER CENT
Tumor visualized	26	65
Atelectasis	27	67

The problem of recognizing early tumor of the lung in the roentgenofluorographic examination is a difficult one. The identification of a primary tumor of the lung is even less easy. A thorough review of the films on the aforementioned 40 patients knowing that they had carcinomatous lungs resulted in identifying only 26, or 65 per cent. That number is greater than the number originally recognized as tumor by the roentgenologist on routine initial examination. The observation of atelectasis was of little more help. Often atelectasis is observed distal to the site of a discrete tumor mass as a result of bronchial obstruction produced by the mass. The finding of atelectasis alone without other evidence of tumor is in itself not diagnostic of tumor of the lung. The x-ray examination, therefore, is valuable as a diagnostic aid primarily in so far as the particular tumor is discretely evident in the lung fields visualized, but it is often helpful as a suggestive indication of pulmonary pathology.

COMMENTS ON TABLE III

Gross recognition of a bronchiogenic tumor and biopsy is only partly successful in the centrally located tumors. The tumor may be anatomically centrally located, but be situated outside the bronchus and thus be unrecognizable through the bronchoscope. The experienced bronchoscopist can readily recognize the presence of a protruding or fungating carcinoma grossly. Biopsy must be utilized to identify the suspicious lesions. In this series 24 of the 26 with positive findings had biopsies. The remaining 2 recognized were diagnosed grossly.

BRONCHIOGENIC CARCINOMA

I. A PATHOLOGIC CLINICAL CORRELATIVE STUDY OF FULL-SIZE MOUNTS FROM OPERATED CARCINOMAS

FRANCIS J. PHILLIPS, M.D., CLAIR E. BASINGER, M.D.* (BY INVITATION), AND
WILLIAM E. ADAMS, M.D.
CHICAGO, ILL.

THE controversy among clinicians and pathologists regarding the pathogenesis of primary carcinoma of the lung stimulated one of us (W.E.A.) to make full-size mounts of representative cross sections of all tumors of the lung removed. This material has been collected from resected cases in this department over a period of nine consecutive years. The study represents at least three-year follow-up on all cases. An attempt has been made to correlate the type of tumor, the site of the tumor, and the size of the tumor with clinical manifestations as demonstrated by history, physical findings, radio fluorographic interpretations, bronchoscopic examination, and exploratory thoracotomy. A cross section of the entire tumor has been studied in detail for variation in type of malignant cells. The type of cell has been studied in its relationship to the site and size of the tumor, the rapidity of growth, direct extension to contiguous tissues, and metastases to other organs. The tumors have been studied for variation of type of cell within the tumor. All these inherent characteristics of the tumors have been studied in relationship to the duration of symptoms and objective manifestations of the growths per se. No attempt has been made to grade the cell malignancy from the microscopic appearance of the cells within the individual types of tumors. The following tables with individual respective discussions give comprehensive evaluation of the correlations included here.

COMMENTS ON TABLE I

The clinical recognition of carcinoma of the lung is often very difficult. The existence of the tumor is not recognized often until the opportune time for favorable operation has passed. The symptoms bringing the patient to the clinic may be very bizarre and apparently unrelated to tumor of the lung. It is not rare for tumor of the lung to be found in routine x-ray examinations of patients being admitted to the hospital for other diseases. At this hospital the incidence of lung tumors of all types for all admissions both to the outpatient clinic and to the hospital is $\frac{1}{10}$ of 1 per cent.¹ In these examinations 4, or 10 per cent, of the aforementioned cases were discovered at the time of routine chest fluoroscopic admission examination for some other disease. Seven of the cases had no physical findings on regular physical examination. Twenty-five, or 62.5 per cent, of the 40 had some degree of hemoptysis. Any streaking of the sputum with blood was classified as hemoptysis. The number having cough and sputum was 32, or 80 per cent. Only 26 of the group had weight loss. This is a finding usually associated with carcinoma in any part of the body. Although 14 of the group

Read at the Twenty-ninth Annual Meeting of The American Association for Thoracic Surgery, New Orleans, La., March 29-31, 1949.

From the Department of Surgery of the University of Chicago. This work was supported in part by grants from the Douglas Smith Foundation for Medical Research and the Illinois Federation of Women's Clubs.

*Work done in conjunction with Senior Thesals.

this figure of the incidence of squamous cells is significant. The "so-called" adenomas or slower growing carcinomas were all centrally located. They represent 10 per cent of the cases of the entire operated series.

Metastases had occurred in 18 of the series or 65 per cent of the total at the time of operation. Within the groups individually considered the incidence of metastases varied from the over-all percentage widely. In adeno- and squamous-cell grouping the 77.7 per cent metastases within the group represents only 17.05 per cent of the metastases of the entire series. This is the highest percentage of any single group. The incidence in the squamous-cell groups was 11, or 27.5 per cent. Two cases with metastases occurred in the adenoma group. One of these patients had continued a relatively normal life for a period of two years in spite of a proved metastasis to the liver found and biopsied during an abdominal operation two years after the primary tumor had been resected in the lung. This patient, however, died of widespread metastases three years later. Cases have



Fig. 1, C.—Photomicrograph of tumor showing pearl formations.

been reported of these tumors remaining intact and apparently dormant for many years and then becoming obviously malignant.⁶ In the 19 cases with evidence of metastases the anatomic site of the primary tumor was peripheral in the lung in 10 and central in 9. All of the patients with metastases from peripherally located tumors are dead. There are only 6 of the patients dead from the group with centrally located tumors. Comparing these numbers with those of the entire series 28, or 70 per cent, of the resected cases were dead at the time of the study. Since 55 per cent of the tumors were centrally located the greater number of deaths has occurred in the patients with centrally located tumors. This figure, however, is obviously misleading since the study of each individual case reveals that a definite number of the deaths was not directly caused by the tumor, but was either operative deaths or deaths from other causes. It is concluded then, that in this study the incidence of metastases is informative, but the mortality rate is not significant to the analytic correlative study itself.



Fig. 1 (A to C).—Unit No. 308221, male, aged 45 years. Squamous-cell peripheral tumor with pearl formation. No metastases. Living.

Fig. 1, A.—Roentgenogram of chest previous to operation.



Fig. 1, B.—Photograph of tumor in cross section.

The size of the tumor appears to have no relation to the incidence of metastases. In fact, the largest tumor resected, No. 256381, had no demonstrable metastases. The patient died of cardiac failure within a short period of time following the operation. It may be significant that this patient had no symptoms or physical findings preoperatively and the tumor had both adeno and squamous cells within its stroma. The data on size of the tumor is of primary interest in demonstrating the size of the individual tumor itself exclusive of tissue reaction around the lesion. It has been observed in this study that a chronic pneumonitis surrounding a tumor may mislead both the roentgenologist and the thoracic surgeon as well in judging the size of the tumor even at the operating table.

COMMENTS ON TABLE VII

The average age of patients in this group was 52.8 years. The average gross dimensions of the tumors were 3.8 cm. in the largest diameter and 2.94 cm. in the shortest diameter. Five of this group are still alive, or 33.3 per cent. One of the 5 who is living came to surgery as an incidental finding of carcinoma. He had had no symptoms or physical evidence of carcinoma. The tumor was larger than the average of the group, but smaller than the average of the entire series.



FIG. 2 (A to C).—Unit No. 10897, male, aged 48 years. Squamous-cell tumor with metastases to brain. Dead.

FIG. 2, A.—Roentgenogram of chest previous to operation.

One of the other patients who is still living had no symptoms, but had had a forty pound weight loss in the previous eleven months. The tumor was as large as the average of the series. The tumors in the patients living were located, one peripherally and four centrally. None have evidence of late metastases. The remainder of the cases had no significant correlative informative data.

Two of this group had had no symptoms or had no physical findings, yet their tumors were well above the average of the group in size. One is still living

TABLE VII. SQUAMOUS CELL WITH PEARLS

UNIT NO.	SITE	AGE (YR.)	SEX	TUMOR SIZE (CM.)	HEMOP-TYSIS (MO.)	PAIN (MO.)	FEVER AND NIGHT SWEATS (MO.)	EMACIATION		COUGH AND SPUTUM (MO.)	METASTASES	PHYSICAL STATUS	
								(LB.)	(MO.)			LIVING AND WELL	DEAD
114754	P	45	M	2.5 by 1.8	18	None	18	20	18	18	Hilar nodes		1
252425	C	56	M	3.8 by 2.0	2	5	2	15	2	6	Left adrenal		1
319201	C	47	M	3.2 by 2.5	8	None	8	None		8	Hilar nodes		1
286505	C	56	M	1.8 by 1.2	12	12	6	14	12	18	None		1
245319	C	56	M	3.0 by 2.0	3	None	None	15	3 yr.	3	None		1
308221	P	45	M	4.0 by 3.2	None	None	None	None		None	None	1	
343491	C	58	M	6.2 by 5.0	None	None	None	40	11	5	None	1	
278917	C	42	M	2.5 by 1.8	15	None	15	25	15	15	Mediastinum		1
341646	C	40	M	5.0 by 2.0	6	None	18	15	6	6	None	1	
334552	C	58	M	6.2 by 5.0	4	None	None	90	18	6	None	1	
334552	C	59	M	5.5 by 3.0	3	9	3	46	8	7	Mediastinum		1
10897	C	48	M	3.3 by 3.2	None	None	None	None		9	Brain		1
323993	P	58	M	0.5 by 0.7	None	None	None	4	2	None	None	1	
370186	C	59	M	9.5 by 4.5	None	None	None	None		12	None	1	
172703	P	57	M	5.0 by 4.5	None	None	None	None		None	None		1
Number of cases		15			Number located centrally	11				Number located peripherally	4		
Oldest patient		54			Metastasized	5				Average age	52.8 yr.		
					Youngest patient	42					Metastasized	1	

COMMENT ON TABLE VIII

The average gross dimension of the tumors in this group was only slightly larger than in the previous group, but the variation of size within the group was less. In this group the peripheral tumors had within their number the



Fig. 3 (A and B).—Unit No. 294639, male, aged 47 years. Squamous-cell peripheral tumor without pearls. Living. No metastases.

Fig. 3, A.—Roentgenogram of chest previous to operation.



Fig. 3, B.—Photomicrograph of section without evidence of pearls.

after five years and one did not survive. One patient, No. 341646, had had mild hemoptysis for only four months, yet he had lost ninety pounds in weight over a period of eighteen months. The only other symptoms were cough and sputum for six months previous to diagnosis.

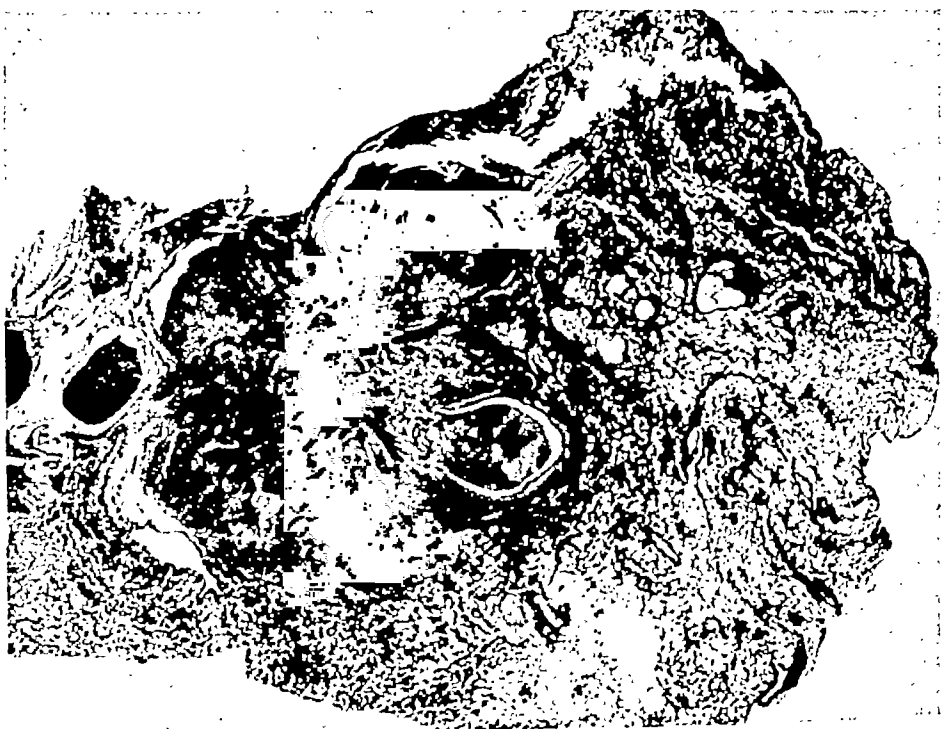


Fig. 2, B.—Photograph of tumor in cross section of resected lung.



Fig. 2, C.—Photomicrograph showing squamous-cell carcinoma with pearl formation.



Fig. 4 (A to D).—Unit No. 35964, male, aged 62 years. Adeno- and squamous-cell peripheral tumor with intermediate transition cell areas.

Fig. 4, A.—Roentgenogram of chest previous to operation.



Fig. 4, B.—Two separate areas with adeno and squamous cells in each.

TABLE VIII. SQUAMOUS CELLS WITHOUT PEARLS

UNIT NO.	SITE	AGE (YR.)	SEX	TUMOR SIZE (CM.)	HEMOP-TYSIS (MO.)	PAIN (MO.)	FEVER AND NIGHT SWEATS (MO.)	EMACIATION		COUGH AND SPUTUM (MO.)	METASTASES	PHYSICAL STATUS	
								LB.	(MO.)			LIVING AND WELL	DEAD
166063	G	42	M	2.0 by 2.0	2	None	2	20	12	2	Subcarinal node	1	
191716	G	58	M	4.5 by 2.5	9	None	2	10		9	Mediastinum		1
246006	P	51	F	7.5 by 3.8	None	None	None	15	2	None	None		1
243218	G	60	M	5.1 by 3.7	2	None	None	25	13	24	None		1
294639	P	47	M	6.6 by 6.9	None	None	None	46	18	18	None	1	
320031	P	44	F	5.1 by 3.7	None	8	None	None		2	None		1
341193	P	54	M	2.0 by 3.0	7	None	7	None		12	Hilar nodes		1
349403	P	64	M	1.5 by 2.2	None	10	None	25	12	10	Hilar nodes		1
360632	P	65	M	1.5 by 2.0	6	None	None	19	6	6	Mediastinum		1
337620	C	64	M	5.0 by 4.4	3 wk.	None	None	None		None	None		1
328415	P	64	M	4.0 by 1.5	3 wk.	3	None	None		12	Hilum and liver		1
240691	C	62	M	1.5 by 3.9	16	16	16	None		16	None	1	
Number of cases				12	Number located centrally		6	Number located peripherally			Metastasized	6	
Oldest patient				65	Youngest patient		42	Average age				57 yr.	

TABLE IX. ADENO AND SQUAMOUS CELLS

UNIT NO.	SITE	AGE (YR.)	SEX	TUMOR SIZE (CM.)	HEMOP-TYSIS	PAIN (MO.)	FEVER AND NIGHT SWEATS	EMACIATION		COUGH AND SPUTUM	METASTASES	PHYSICAL STATUS	
								(LB.)	(MO.)			LIVING AND WELL	DEAD
233314	P	62	M	7.0 by 5.0	1 yr.	None	None	25	12	12 mo.	Right kidney	1	
234309	P	50	M	5.0 by 6.0	None	2	None	16	2	12 mo.	Chest wall		1
256381	P	55	M	9.5 by 7.5	None	None	None	40	4	None	None		1
240063	P	69	F	2.0 by 3.0	None	None	None	None		3 yr.	Mediastinum		1
359364	P	62	M	6.2 by 4.0	None	None	None	7	1	None	Mediastinum		1
321365	P	57	M	6.2 by 3.0	2 wk.	None	None	35	3	2 wk.	Pericardium		1
299507	P	44	M	4.2 by 3.8	None	7	1 wk.	20	8	12 mo.	Mediastinum		1
340660	P	57	M	3.0 by 2.5	None	None	12 mo.	15	12	10 mo.	None	1	
342015	C	50	M	4.2 by 3.5	12 mo.	12	12 mo.	35	12	12 mo.	Pericardium	1	
Number of cases				9	Number located centrally		1	Number located peripherally			Metastasized	8	
Oldest patient				69 yr.	Youngest patient		44	Average age				55.7 yr.	

TABLE X. ADENOMATA OR SLOWER GROWING CARCINOMA

UNIT NO.	SITE	AGE (YR.)	SEX	TUMOR SIZE (CM.)	HEMOPHY- TIC	PAIN	FEVER AND NIGHT SWEATS	EXAMINATION		COUGH AND SPUTUM	METASTASES	PHYSICAL STATUS	
												LIVING AND WELL	DEAD
226523	C	33	F	1.6 by 1.8	4 mo.	None	None	20	2 yr.	5 yr.	None	1	
243282	C	42	F	3.5 by 1.5	None	11 mo.	None	30	18 mo.	18 mo.	Bone marrow		1
165902	C	24	M	4.0 by 4.0	6 yr.	None	8 yr.	18	5 mo.	10 yr.	None	1	
232383	C	34	F	2.0 by 2.0	10 yr.	None	None	10	10 yr.	10 yr.	Liver and brain		1
Number of cases				4	Number located centrally		4	Number located peripherally				0	
Oldest patient				62	Metastases		2	Average age				38.2 yr.	
					Youngest patient		24						

larger sized lesions. In the previous group one patient with a centrally located tumor had the largest sized lesion and had as the only symptom cough and sputum for a period of one year previous to diagnosis. There was no signifi-



Fig. 4, C.—High power photomicrograph showing transition between two types of carcinoma cells.



Fig. 4, D.—Low power photomicrograph of area with adeno- and squamous-cell structures contiguous.

the spine. Another one of the group, No. 232383, is of particular interest because of long treatment and clinical course. The tumor was first discovered in 1933. The patient was observed for some period of time. In 1935, she was given



Fig. 5, B.—Photomicrograph of primary tumor in lung.

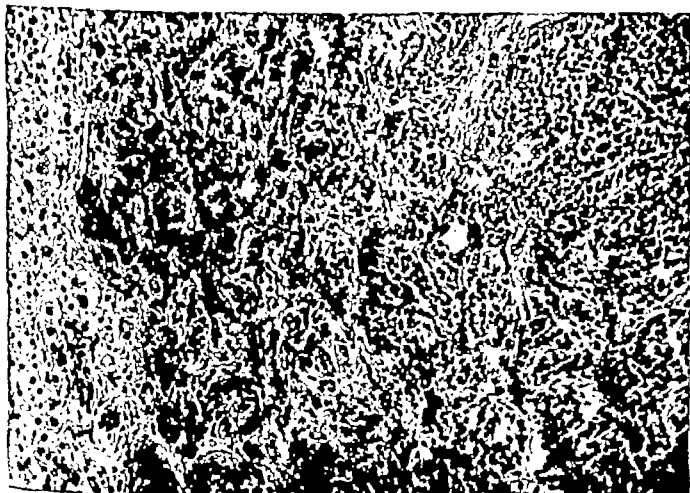


Fig. 5, C.—Photomicrograph of liver metastasis.

cance as to variation in the metastatic incidence in the two groups. Two of the patients in this group had had hemoptysis for only three weeks previous to diagnosis. One of the patients, No. 328415, had metastases to the liver and to the lung hilum, yet he had no weight loss. Neither patient survived. One of the three patients, No. 294639, who is still living, had had a weight loss of forty-six pounds over a period of eighteen months. He had had no pain. His tumor was classified as peripheral.

COMMENT ON TABLE IX

This group had the greatest number of cases with the tumor in the periphery. These were the fewest with hemoptysis. The weight loss was somewhat greater in this group. All but 2 of the cases had metastases either to contiguous organs or in one case to the kidney, No. 233314. This group carried the highest mortality on a percentage basis. The average size of the tumors in their greatest dimensions was 5.3 by 3.2 cm. The largest tumor occurring in the entire series was in this group, No. 256381. This tumor had not metastasized. The patient did not survive. This group had the greatest number of metastases to the pericardium. The only metastases to the chest wall occurred in this group. One case in this group, No. 359364, is especially interesting in that the tumor was peripheral with both adeno- and squamous-cell areas adjacent to each other with an area of transition cells in between as illustrated in Fig. 4.



Fig. 5 (A to C).—Unit No. 232383, female, aged 24 years. Adenoma or slow growing carcinoma of lung with metastases to liver and brain. Dead.

Fig. 5, A.—Roentgenogram of chest previous to operation.

COMMENT ON TABLE X

This group of slower growing carcinoma represents 10 per cent of the cases in the study. One of the group had an early metastasis to the bone marrow of

tases came from the adeno-squamous type. Only 2 of these 6 had pain. Only 3 had hemoptysis. Two of the 3 had hemoptysis for one year or more. This would appear to indicate the presence of a growing tumor for a long period of time. One had hemoptysis only two weeks and yet that tumor was one of the largest of all types resected.

The potential malignancy of the so-called adenoma is further substantiated in 2 of the 4 cases presented. The death rate and metastasis rate were both 50 per cent. One metastasized to the brain and one to the spine, thus following common carcinoma metastatic patterns. Although there may be some plausible debate as to the cell origin and growth rate there is little doubt of the ultimate course of the tumor. Our cases and cases reported by other surgeons previously cited in this paper give convincing proof of the malignant nature of the so-called adenoma. One of us previously ascribed the term "malignant adenoma" to this type of tumor.¹⁰ Now it appears that it may be prognostically sagacious to consider them carcinoma in general and accord them the serious respect given carcinoma of the lung. The ineffectiveness of x-ray therapy is demonstrated by our patient, No. 232383, who had massive irradiation yet she died of metastasis to the brain and liver. One patient with a squamous-cell carcinoma with pearl formation had x-ray evidence of a large tumor of the chest for a period of three years, yet there were no symptoms. He died of cardiac complications a few weeks after resection. Complete post-mortem examination revealed no metastases. Comparing the adenoma, so-called, with this proved case of carcinoma, it is difficult to ascribe less serious significance to the adenoma groups.

The time of diagnosis of the lung tumor appears to be of more prognostic significance than the type of cell found in the tumor or its location in the lung.

CONCLUSIONS

1. A study of 40 resected carcinomas of the lung has been made.
2. Squamous-cell and adeno-cell carcinoma have been demonstrated immediately adjacent within the same tumor mass.
3. Squamous-cell carcinomas with and without pearl formation were found in 36 individual tumors. No pure adeno type carcinoma cell tumors were found.
4. Two proved cases of metastasis of a so-called adenoma were studied and discussed.
5. No correlation between type of tumor cell or location of tumor within the lung and the prognosis was demonstrable in this study.
6. Lung carcinoma occurred almost six times more frequently in men than in women.

REFERENCES

1. Block, Robert G., and Tucker, W. B.: The Indispensability of Routine X-ray Examinations of the Chest in a General Clinic, *Am. Rev. Tuberc.* 50: 405, 1944.
2. Gibbon, John H., Jr., Clerg, Louis H., Herbut, Peter A., and De Turek, John J.: The Diagnosis and Operability of Bronchiogenic Carcinoma, *J. THORACIC SURG.* 17: 419, 1948.
3. Block, Robert G., Adams, W. E., Thornton, Thomas F., and Bryant, Edmond J.: Difficulties in the Differential Diagnosis of Bronchiogenic Carcinoma, *J. THORACIC SURG.* 14: 83, 1945.

x-ray therapy to the tumor through the chest wall. The x-ray evidence of the tumor disappeared. However, the mass reappeared approximately eight years later. At that time, Oct. 14, 1943, a right bilobectomy was performed. Post-operative course was uneventful. She was apparently relieved of lung symptoms. Two years later, or 1945, a pelvic operation was performed through the abdomen. On exploration nodules were demonstrable in the liver. Microscopic section of these nodules revealed a morphologic picture like that of the tumor removed from the lung two years previously. She survived until June, 1948, at which time she died with generalized metastases including the brain. These 4 cases were included in this series because of the similarity of symptoms to those of the more rapidly growing carcinomas. The main difference in the two types of tumors appears to be in the rate of growth.

DISCUSSION

This is believed to be the first such correlative study made of cross sections of resected lung carcinoma. We believe that the study is peculiar in that it demonstrates rather unequivocally that many of the former concepts regarding the growth, metastases, and microscopic characteristics of carcinoma are no longer tenable in dealing with these tumors of the lung.

The evidence presented in this paper that any combination of cell types may exist in the same tumor belies many of the previous beliefs that a particular cell type has a predilection for some specific area of lung tissue. This study of entire sections of tumors after resection has been very illuminating. Squamous cells with pearls were not found in the same tumor with adenocarcinomatous type cells. This may be indicative of growth phases, but there is no good proof that coincidence is not the correct explanation. This general finding is in close agreement with the concept of Graham and others that these tumors are really all carcinoma.^{8, 9} Their unpredictable operability is again demonstrated. It is of special interest that we found no pure adenocarcinoma in any of these cases. One case had both adeno and squamous cells in close proximity to each other with a physical merging of one into the other (Fig. 4).

The finding of a greater number of metastases in the peripheral tumors is again an indication of failure of early recognition of the tumor. Proper clinical evaluation of mass chest x-ray examinations will reveal these apparent symptomless carcinomas in their early phases before metastasis or direct extension has taken place. The higher mortality rate of the peripheral tumors may well be attributed to late recognition and consequent more or less heroic surgical treatment. That the peripheral tumors are more malignant or are more consistently one type of tumor is a theory that is not substantiated in our series. Our study appears to indicate that the centrally located tumor has the lower metastasis rate and the lower death rate. We believe, however, that these findings stem from the fact that the central tumor presents symptoms earlier and is consequently removed before sufficient time has elapsed to allow metastasis to take place, and not because the peripheral tumor is more malignant. Ten of the 18 peripheral cases were of a squamous-cell type and 8 were adeno-squamous-cell type. Yet 11 of the 18 metastasized. Out of that 11, 6, or 54.5 per cent, of the metas-

BRONCHIOGENIC CARCINOMA

II. THE CORRELATION OF PATHOLOGIC CHARACTERISTICS AND CLINICAL MANIFESTATION IN UNRESECTABLE AND UNEXPLORED POST-MORTEM LUNG TUMORS

GEORGE M. BOGARDUS, M.D. (BY INVITATION), KALISPELL, MONT., AND WILLIAM E.
ADAMS, M.D., AND FRANCIS J. PHILLIPS, M.D., CHICAGO, ILL.

GRAHAM¹ has emphasized the need for continued study of carcinoma of the lung in order to extend knowledge and to improve the rate of cure. A study of the resected tumors of the lung has been made in this clinic by Phillips, Basinger, and Adams.² As a supplement to the paper by Phillips and associates this paper is concerned with information obtained from a study of carcinoma of the lung which was explored but found to be nonresectable, and carcinoma of the lung not operated upon but found at autopsy.

Adler³ stated that perhaps Morgagni (1682-1772) was the first to publish the results of autopsies that might be diagnosed as cancerous. Following the fundamental discovery of Schwann, histology assumed a dominant role in pathology. According to Ewing,⁴ the earliest microscopic studies of lung tumors were by Landhans, Marchiafava, and Malassez.

From a histologic standpoint, Adler assigned the majority of cases to the carcinoma group, and it is probable that present day pathologists would limit even further his group of sarcomas in which were the spindle and round cell forms now considered by some to be variants of carcinoma.⁵ Under carcinoma simplex he included squamous and alveolar types. A second large group was composed of cylindric cells. He makes no mention of behavior of the tumor with respect to the cell type.

In Table I is shown clinical data pertinent to the age, sex, and symptomatology of the explored but nonresected group. It is apparent that men outnumber women about 5 to 1. The average age was 52 years and symptoms of cough, weight loss, sputum, chest pain, dyspnea, and hemoptysis were predominant in this group. It might be pertinent to note that the symptoms commonly associated with carcinoma of the lung occur in the advanced stages of the disease. Without wishing to discourage further investigation and attempts at therapy in this group, however, there is evidence that these tumors may be detected prior to the onset of symptoms by means of fluoroscopy and x-ray examination. This has been ably pointed out by Bloch and Tucker⁶ and by Adams.⁷

Several studies of the cytology and behavior of carcinoma of the lung have been made in recent years, for example, Boyd,⁸ Fried,⁹ Karsner,¹⁰ Weller,¹¹ Gebauer,¹² and Koletsky.¹³ There has been relatively little emphasis upon the correlation of clinical behavior with the cell types. Notable among these attempts, however, was the work of Koletsky¹³ who concluded that "the histologic

From the Department of Surgery of the University of Chicago.

This work was supported in part by a grant from the Illinois Federation of Women's Clubs.

Read at the Twenty-ninth Annual Meeting of The American Association for Thoracic Surgery, New Orleans, La., March 29-31, 1945.

4. Brewer, Lyman A., III, Jones, Wilfred M. G., and Dolley, Frank S.: Nonmalignant Intrathoracic Lesions Simulating Bronchiogenic Carcinoma, *J. THORACIC SURG.* 17: 439, 1948.
5. Tuttle, W. M., and Womack, Nathan A.: Bronchogenic Carcinoma: A Classification in Relation to Treatment and Prognosis, *J. THORACIC SURG.* 4: 125, 1934.
6. Graham, Evarts A., and Womack, Nathan A.: The Problem of the So-called Bronchial Adenoma, *J. THORACIC SURG.* 14: 106, 1945.
7. Graham, Evarts A.: Bronchiogenic Carcinoma, *Surg., Gynec. & Obst.* 88: 129-131, 1949.
8. Womack, N. A., and Graham, E. A.: Mixed Tumors of the Lung, *Arch. Path.* 26: 165, 1938.
9. Rabinovitch, Jacob, Hochberg, Lew A., and Lederer, Max.: Primary (Bronchiogenic) Carcinoma of the Lung, *J. THORACIC SURG.* 9: 332, 1940.
10. Adams, W. E., Steiner, P. E., and Block, R. G.: Malignant Adenoma of Lung Carcinoma-Like Tumors With Long Clinical Course, *Surgery* 11: 503-526, 1942.

DISCUSSION

DR. JEROME HEAD, Chicago.—A great many years ago I had the opportunity of studying at the Mayo Clinic cell type and degree of malignancy, and Dr. McCarty, who was a good deal of an iconoclast and a tremendously imaginative person, was interested in asking why in one individual you have a rapidly growing tumor and in another individual a slowly growing tumor. He was interested in the resistance of the body to carcinoma. I think we have a tendency to approach the carcinoma problem from the wrong angle, considering these tumors as entirely isolated from the host, and as entirely uninfluenced by the host. It is quite conceivable to me that we all, at one time or another, have many carcinomas but that we are able to overcome them; we have a high resistance to the efforts of the cells to become autonomous. Perhaps the logical approach to the carcinoma problem is to increase the resistance of people to carcinoma rather than attempt to cure the occasional case where the carcinoma gets out of hand. I think, therefore, that when we talk about types of carcinoma, types of cells, grades of carcinoma, rapidity of growth, we should ask why does this carcinoma grow rapidly in this person, and consider the host as an important factor in the carcinoma problem.

DR. F. J. PHILLIPS.—I am very sorry that Dr. Graham is not here to discuss this paper. It was the stimulation of his studies and interpretation that stimulated Dr. Adams to start this work originally. We appreciate Dr. Head's speculations and discussion. Certainly in the light of what has been said about another type of carcinoma, adenomatosis, there may well be something to the individual's resistance to the tumor. There are others of us who feel somewhat the same way. Why, we do not know specifically.

Further discussion of the conclusions is not indicated. We have presented these studies as evidence of what may be found if the entire tumor is studied in cross section, and we believe that the sections speak for themselves.

and 4 were undifferentiated, while 1 was reported as a basal cell tumor and 1 was a mixed form. The remainder had been bronchoscoped by outside doctors and their slides were not available for study. The biopsies removed at thoracotomy were found to be undifferentiated in 11, nonkeratinizing squamous in 5, a mixed type in 4, and adenocarcinoma in 1.

TABLE II. PATHOLOGIC FEATURES OF CARCINOMA OF THE LUNG EXPLORED BUT NOT RESECTED

Structures invaded by tumor	
Hilar and mediastinal nodes	16
Chest wall	12
Aorta	5
Heart	5
Trachea	2
Bronchoscopy	
Negative	20
Positive	19
Squamous without pearls	
Undifferentiated	11
Outside report	4
Basal	2
Mixed form	1
Surgical pathology	
Undifferentiated	16
Squamous without pearls	14
Mixed	4
Adenocarcinoma	2
Type of tumor with respect to location of biopsy	
Mediastinum	
Squamous without pearls	6
Undifferentiated	8
Mixed form	1
Adenocarcinoma	2
Chest wall	
Squamous without pearls	3
Undifferentiated	7
Adenocarcinoma	1
Mixed forms	3

There is some evidence to suggest that in this group the undifferentiated tumors tended to occur at a greater distance from the hilum. The one adenocarcinoma chiefly involved the mediastinum and the aorta, while of the mixed types, one spread mainly to the periphery and one was located chiefly at the hilum. The mixed forms (containing both squamous cells and adenocarcinoma) are worthy of comment in that they demonstrate that for really reliable diagnosis and study, large sections are indispensable. That different portions of the same tumor reveal strikingly different histologic patterns is well brought out by Phillips and co-workers.*

This nonresected group of cases demonstrate a somewhat different ratio among the commonly accepted classifications of adenocarcinoma, squamous carcinoma, and undifferentiated carcinoma. In this group there was but one adenocarcinoma. This is contrary to the usually reported frequency of adenocarcinoma which ordinarily comprises between one-third and one-fourth of all lung carcinomas. It will also be noted that keratinizing squamous tumors were absent. The majority of the tumors were either nonkeratinizing squamous or undifferentiated tumors.

classification of primary carcinoma of the lung may be correlated with essential differences in the growth, dissemination and prognosis of each tumor. The small cell carcinoma is a highly malignant tumor which offers a poor prognosis. Usually primary at the hilus of the lung, it readily invades the postero-superior mediastinum and metastasizes extensively, with special tendency to widespread extension through the lymphatic system. The tumor occurs in younger persons, and there is a short rapid course without notable remission. The squamous carcinoma is slowly growing, locally invasive, and relatively non-metastasizing growth accompanied by infection, necrosis and cavity formation. Involvement of lymph nodes other than the regional and tracheobronchial nodes is infrequent. This type of carcinoma, particularly a growth involving the hyparterial bronchi of the lower lobe offers the most favorable prognosis for complete resection. In cases of adenocarcinoma surgical intervention is less favorable, since the tumor, while locally invasive, shows more frequent and more extensive lymph node involvement and metastasizes injuriously by way of the blood stream." That this opinion is not unanimous at present is shown by two recent articles in the JOURNAL in which there seemed to be disagreement as to the value of the cytologic data in estimating prognosis.^{14, 15}

TABLE I. THIRTY-SIX CARCINOMAS OF THE LUNG EXPLORED BUT NOT RESECTED

Men	30
Women	6
Average age (yr.)	52
Symptoms	
Cough	32
Weight loss	31
Sputum	27
Pain	27
Dyspnea	23
Hemoptysis	20
Anorexia	16
Fever	12
Night sweats	12
Average of longest symptom (mo.)	7
Average weight loss (mo.)	21

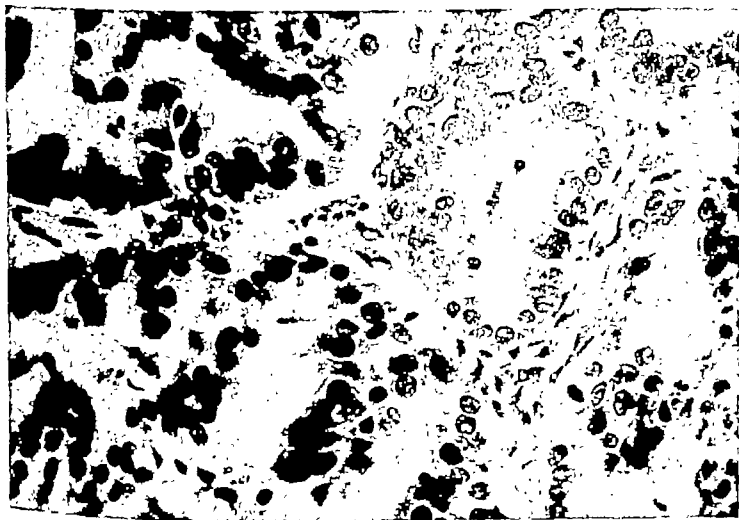
Adams¹⁴ reported that "the cell type of carcinoma found by microscopic examination is the most important single consideration in estimation of prognosis after a resection. If the growth is undifferentiated or oat-cell carcinoma in type, less than a year of life may be expected. Of 15 patients now living and well beyond one year, 12 had epidermoid carcinoma, 2 had adenocarcinoma, and 1 undifferentiated carcinoma." In contrast with this statement is the report of Neuhof and Aufses¹⁵ that "the microscopic features of cancer of the lung bear no significant relation to the ultimate prognosis."

Table II summarizes some of the pathologic information on our group of nonresected cases. It will be seen that the majority of these tumors fall into two groups, the undifferentiated, and the nonkeratinizing squamous variety. A much smaller group is composed of mixed forms containing more than one cell type, for example, both adenocarcinoma and nonkeratinizing squamous may be present. Of the bronchoscopic specimens, 9 were nonkeratinizing squamous

There were 36 autopsied but not explored patients with carcinoma of the lung. The average age of this group was 56 years, and 29 of the 36 were men. The correct diagnosis was made in one-half of the cases prior to autopsy. About



A.

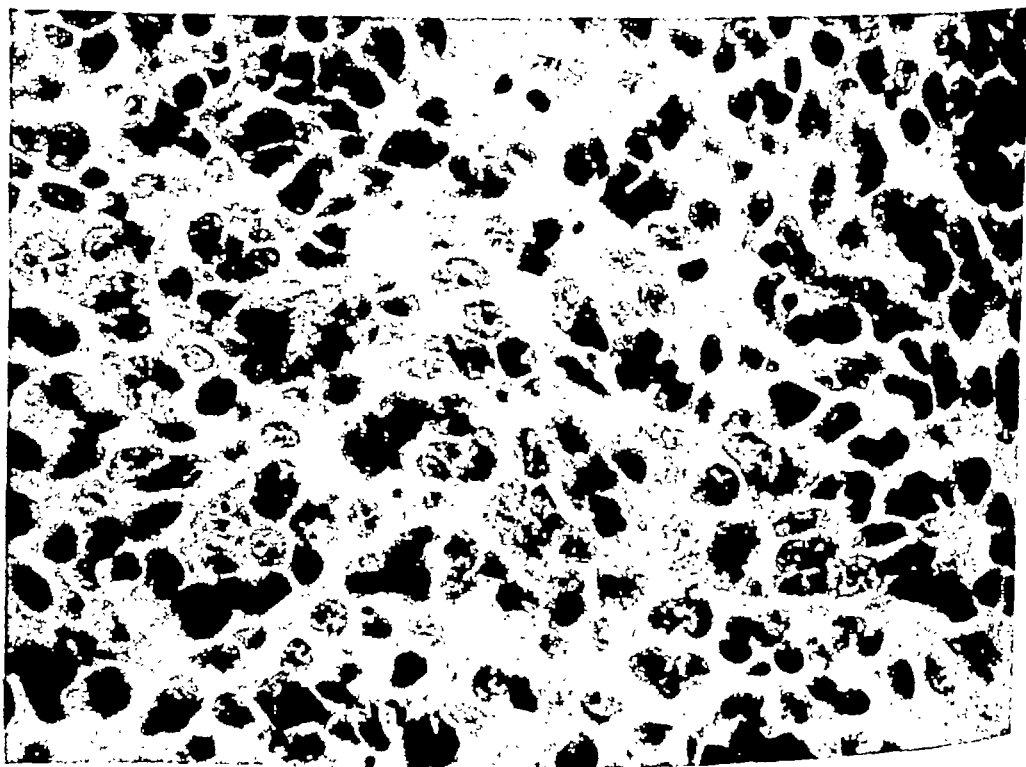


B.

FIG. 2.—X-ray therapy had been given without a proved diagnosis. A, X-ray appearance prior to death; B, microscopic appearance of the adenocarcinoma which arose in an old tuberculous scar.



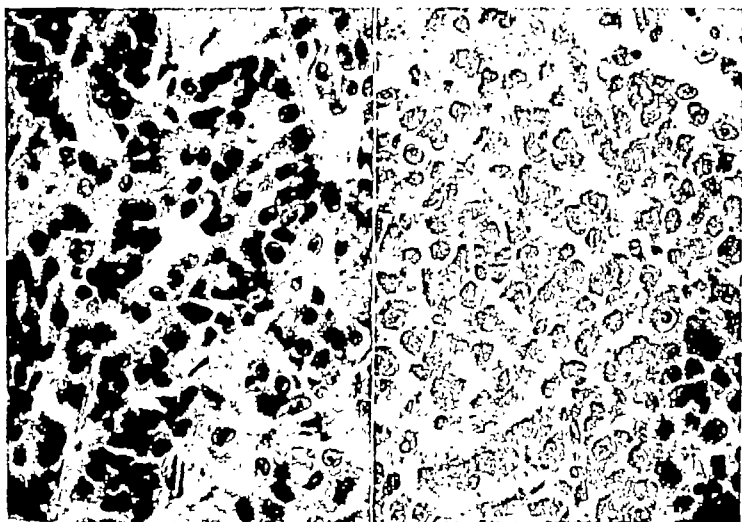
A.



B.

Fig. 1.—A, Roentgenogram of a patient who had an undifferentiated carcinoma of the lung;
B, microscopic appearance of this patient.

remote metastases. Briefly, the specific sites for spread of the tumor, in those correctly diagnosed, were: pleura, 10; chest wall, 5; the brain in 2, and the mandible in 1.



A.

B.



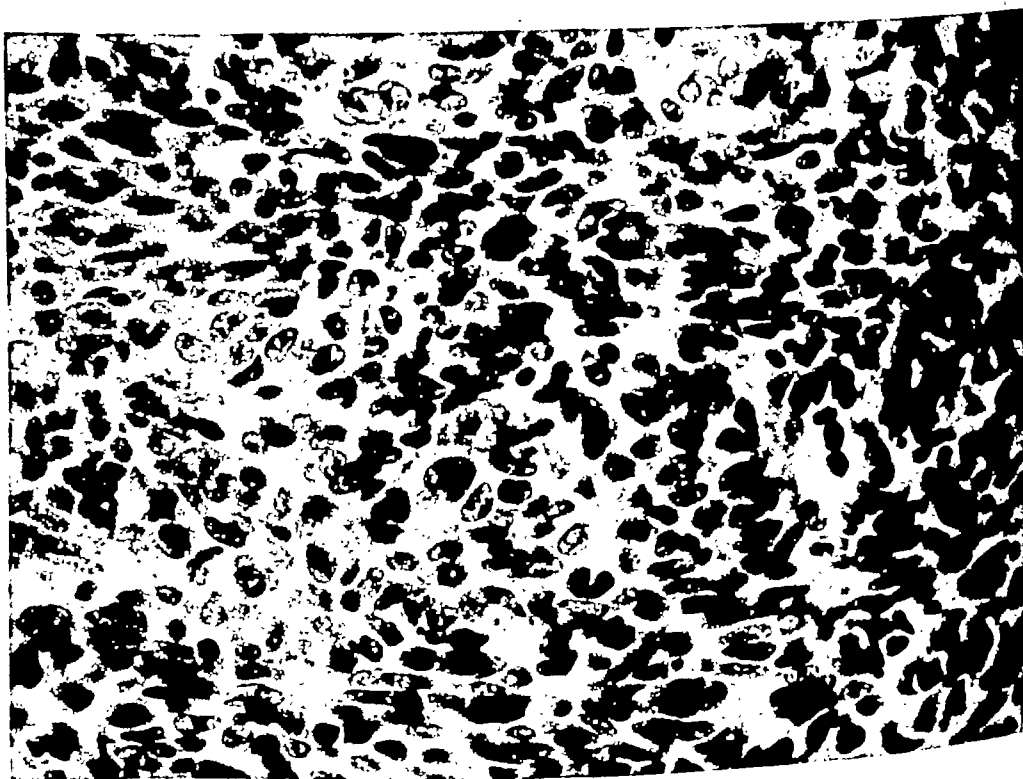
C.

Fig. 4.—Shows two cell types in the same tumor. A is from the primary site, being an adenocarcinoma and B is an undifferentiated metastasis. C, X-ray appearance of the chest in the preceding case.

the same ratio held true in Koletsky's series of 100, the correct diagnosis having been made ante-mortem in 56 of the cases. All of our 18 diagnosed cases were unsuitable for operation because of extensive pleural spread, or because of more



A.



B.

Fig. 3.—A, Roentgenogram of an unexplored patient whose tumor area arose in the right main bronchus; B, microscopic appearance of undifferentiated tumor.

PATHOLOGY

Of the 36 autopsied carcinomas, 15 were adenocarcinoma, 14 were undifferentiated, 6 were squamous, and 1 was a combination of adenocarcinoma and nonkeratinizing squamous. The relationship between cell type, site of origin, and the tendency to either predominantly mediastinal invasion or widespread metastases is shown in Table V. It will be seen that in this rather small group the squamous variety tended to occur nearer the hilum whereas the more peripheral group was largely adenocarcinomas or undifferentiated tumors. There is a tendency in this group for the peripherally located undifferentiated tumors and for the adenocarcinomas to metastasize widely, whereas the centrally located squamous variety tend to invade locally and have a limited tendency to metastasize outside the chest. This impression conforms to previous reports by Koletsky¹¹ and Gebauer.¹²

TABLE V. THIRTY-SIX CARCINOMAS IN THE AUTOPSY FILE NOT OPERATED

		PERIPHERAL	CENTRAL	UNCERTAIN
Adenocarcinomas	15	9	4	2
Undifferentiated	14	6	7	1
Squamous	6	1	4	1
Mixed	1 (secondary bronchus)			
Widespread extrathoracic metastases in 18				
Adenocarcinoma		6	3	
Undifferentiated		5	3	
Squamous			1	
Extensive mediastinal spread in 11				
Adenocarcinoma		2	1	
Undifferentiated		2	2	
Squamous		1	3	

Some of the microscopic features may be discussed. There were three chief groups, as indicated above, adenocarcinoma, squamous-cell carcinoma, and the undifferentiated. Occasionally, bizarre forms are seen and not infrequently tumors composed of both adenocarcinomas and nonkeratinizing squamous tumors are found. The histology of the adenocarcinomas consists of abnormal acinous formation by irregular large or small columnar cells. The individual cells vary in their degree of differentiations, but usually present no problem in recognition. Mucous production is not a constant feature.

The squamous tumors are composed of either the keratinizing or nonkeratinizing group. The former is readily recognized by the flat sheets of polygonal cells, and the tendency to pearl formation. Prickle-cell formations are sometimes recognizable. The nonkeratinizing group is made up of large sheets of polyhedral cells, at times showing prickle formation, and no tendency to acinous development.

The third large group is the undifferentiated or "oat-cell" tumor. These probably were mistaken in earlier days for sarcoma. The cells are spindle or oval shaped, growing often in bundles and having a somewhat sarcomatous appearance. Ochsner⁵ has commented on the different types which the relatively undifferentiated tumors may assume. His recent article contained excellent illustrations of these forms. A smaller group makes up a list of bizarre tumors

In the group of 18 diagnosed as primary lung tumors, the average age was 56 years, and 14 of the 18 were men. Nearly all of these patients had symptoms referable to the chest such as cough, chest pain, sputum, or dyspnea. Weight loss was also an important finding. The average duration of symptoms with respect to tumor histology was as follows: adenocarcinomas, eight months; squamous, four months; and the 1 mixed type had produced symptoms for 1 month.

Bronchoscopy was used in only 6 of these cases. In one, the examination was inconclusive. In 3, there was gross evidence of tumor, but no microscopic sections were made. Two of the 6 had microscopic sections; 1 of these showed squamous cells and the other was undifferentiated. The one which bronchoscopy reported as a squamous tumor really was undifferentiated according to the autopsy findings. The other case, that of D. H., had a bronchoscopic report of squamous-cell carcinoma of bronchus and was, at autopsy a squamous tumor.

Biopsies are frequently useful and may be obtained from various areas. Table III compares the result of biopsy in these cases with the autopsy findings.

TABLE III. A COMPARISON OF SURGICAL BIOPSIES WITH AUTOPSY DIAGNOSES TO INDICATE THE VARIABILITY OF THE CELL TYPE IN THE SAME PATIENT

CASE	SITE	BIOPSY REPORT	AUTOPSY REPORT
1	Cervical nodes	Squamous	Adenocarcinoma
2	Mandible	Squamous	Mixed
3	Larynx	Squamous	Squamous
4	Chest wall	Undifferentiated	Adenocarcinoma
5	Ilium	Undifferentiated	Undifferentiated

It is apparent that not too much confidence can be placed in the histologic report of the biopsied specimens. Many times, however, a careful physical examination will reveal nodes or lumps which can be biopsied. If positive for carcinoma, such information is helpful in establishing a diagnosis. The authors have obtained positive biopsies from such places as the posterior chest wall, axilla, lateral chest wall, skull, extremities, cervical nodes, and maxilla.

TABLE IV. THE LOBAR DISTRIBUTION OF THESE TUMORS WAS AS FOLLOWS

Right upper lobe	5	Left upper lobe	6
Right main bronchus	5	Left lower lobe	5
Right lower lobe	4	Undetermined	3
Middle lobe	2	Left main branches	

Eighteen of the autopsy group were undiagnosed prior to death. Four of these were tiny incidental tumors occurring during the course of other diseases. Of the remaining 14, the clinical manifestations were caused by the bronchiogenic tumor, but were misinterpreted. Six of the 14 undiagnosed tumors were from autopsies on patients from outside institutions. The eight errors in diagnosis at this hospital consisted of mistaking the lesion for aneurysm, lymphoma, tuberculosis, lung abscess, and brain tumor. The histology of the undiagnosed group was as follows: undifferentiated 8, adenocarcinoma 7, and squamous 3. Four of this group were women. The average age of the 18 was 56 years. The lobar distribution of these tumors is seen in Table IV.

THE MANAGEMENT OF CHYLOTHORAX

RICHARD H. MEADE, JR., M.D., GRAND RAPIDS, MICH., JEROME R. HEAD, M.D., CHICAGO, ILL., AND (BY INVITATION) CHESTER W. MOEN, M.D., BOSTON, MASS.

THE management of chylothorax was mainly a subject of academic interest until Lampson,² in 1946, demonstrated that the condition could be cured by ligation of the thoracic duct in the lower chest. Prior to this, only fifty-eight cases of traumatic chylothorax had been reported, and fewer reports of chylothorax due to other causes had appeared in the literature. According to Zesas,¹² the first description of a case of traumatic chylothorax was made by Bartolet in 1633. As about one-half of the cases of chylothorax have been associated with fatal diseases, and as they are rare, there has been little enthusiasm for working out a satisfactory method of treatment. However, the other one-half of the cases of chylothorax are caused by direct or indirect violence, or by injury during an intrathoracic operation. That it may occur following seemingly trivial trauma has also been known for a long time. The mortality from chylothorax, from all causes, has been put at 50 per cent by many writers. Recently such excellent presentations of the subject have been published by Lampson,² by Baldrige and Lewis,¹ and by Hodge and Bridges,⁴ that any further survey of the whole subject would be superfluous. However, it is worth while to discuss further the subject of treatment, and to present three new cases in which different forms of treatment were successfully used.

Soon after Lampson submitted his article on traumatic chylothorax cured by ligation of the thoracic duct for the 1947 program of The American Association for Thoracic Surgery, we were asked to see a patient with chylothorax. She was 24 years of age, and had fallen down cellar steps, without apparent injury, three months before developing symptoms of pleural effusion. After nine weeks of study for a possible cause for chylothorax, which included a test of x-ray therapy, it was decided that it was due to the fall. Protein balance was maintained by means of a high protein diet and repeated infusions of plasma, and the pleural cavity was frequently emptied of its chyle. She was operated upon, and the thoracic duct was ligated just above the diaphragm. Although the approach to the duct was not as easy as described by Lampson, prompt recovery made this method of treatment seem to be the logical one (Figs. 1, 2, and 3). A short while after this patient was discharged from the hospital another patient, a woman 58 years of age, with chylothorax, was referred to us. After another exhaustive study to rule out a suspected malignancy, and after repeated aspirations of the chyle and partial replacement of the lost protein by plasma infusions, she was operated upon. At this operation a section of the thoracic duct was supposedly resected a short distance above the diaphragm on the right, but on histologic examination it was seen to be splanchnic nerve.

Read at the Twenty-ninth Annual Meeting of The American Association for Thoracic Surgery, New Orleans, La., March 29-31, 1949.

containing the so-called adenoma, which we believe to be malignant. Phillips² emphasized that mixed forms having more than one cell type may occur in the same tumor. Representative slides along with the roentgenograms of some of the various tumors are shown in Fig. 1.

Of the 36 autopsied, but not explored, lung carcinomas, 15 were adenocarcinomas, 14 were undifferentiated, 6 were squamous, and 1 was a mixed form. Four of the 6 squamous carcinomas were keratinizing while 2 were nonkeratinizing. In these cases, the adenocarcinomas and the undifferentiated, or "oat-cell" tumors, tended to lie toward the periphery and spread not only to mediastinal structures, but also to distant organs via the blood stream. The squamous variety tended to occupy a more central location and spread chiefly through adjacent mediastinal structures.

SUMMARY

The pathologic type and the clinical behavior of explored but nonresected lung carcinomas, and the lung carcinomas which came to autopsy without exploration have been compared with a group of resected tumors of the lung. It is our impression that such a study sheds some light on the behavior of these tumors. We feel that there is some correlation of cell types with clinical course. Of course, any far-advanced tumor will be inoperable. The tumors which appear to be less amenable to surgical therapy are largely the poorly differentiated ones and the adenocarcinomas. This impression is derived from the fact that the autopsied and the explored but not resected tumors were composed of 30 undifferentiated tumors, 17 adenocarcinomas, 16 nonkeratinizing squamous, 4 keratinizing squamous, and 5 mixed forms containing more than one all type. In contrast to this, Phillips and associates² found that all of their resected tumors were squamous cell in part or entirety.

REFERENCES

1. Lindskog, G. E.: *Ann. Surg.* 124: 667, 1946 (Discussion).
2. Phillips, F., Basinger, Clair, and Adams, W. E.: *J. THORACIC SURG.* 19: 680, 1950.
3. Adler, Isaac: *Primary Malignant Growth of Lungs and Bronchi*, London, 1912, Longmans, Green & Co.
4. Ewing, James: *Neoplastic Diseases*, ed. 4, Philadelphia, 1942, W. B. Saunders Company.
5. Ochsner, Alton, and associates: *J. THORACIC SURG.* 17: 573, 1948.
6. Bloch, R. G., and Tucker, W. B.: *Am. Rev. Tuberc.* 50: 405, 1944.
7. Adams, W. E.: *Clinics* 4: 1035-1054, 1945.
8. Boyd, William: *Canad. M. A. J.* 23: 210, 1930.
9. Fried, B. M.: *Bronchiogenic Carcinoma*, Baltimore, 1932, Williams & Wilkins Company.
10. Karsner, Howard: *Human Pathology*, ed. 6, Philadelphia, 1943, J. B. Lippincott Company.
11. Weller, C.: *Arch. Path.* 7: 478, 1929.
12. Gebauer, P. W.: *J. THORACIC SURG.* 10: 373, 1941.
13. Koletsky, S.: *Arch. Int. Med.* 62: 636, 1938.
14. Adams, R.: *J. THORACIC SURG.* 17: 306, 1948.
15. Neuhoef, H., and Aufses, A. H.: *J. THORACIC SURG.* 17: 297, 1948.

With this information, and with the appearance of chyle in the opposite pleural cavity, she was operated upon again in an effort to ligate the leaking duct. In the history it had been learned that she had fallen down some steps six months previously without apparently being hurt. Because of our first case we thought that this fall was the etiological factor in this case. At the first operation nothing abnormal could be found except the chyle in the pleural cavity. The second operation was done through the same right side because it was thought that the leak was on this side but that the chyle had not been able to re-enter this pleural cavity as a result of its obliteration by postoperative adherence of the pleural surfaces. Closed drainage of the right pleural cavity had been maintained for two days after the operation. At the second operation it was seen that all of the pleural space had been obliterated by the re-expanded lung. An egg-nog had been given to the patient two hours before the operation in order to make the thoracic duct more prominent. After an exhaustive search, chyle was seen leaking from a small duct. This vessel was ligated, but it was obviously not the only channel through which the chyle was flowing. After further search failed to reveal another duct, the space between the vertebral column and the diaphragm was packed with Oxyeel gauze in hope of blocking any other pathway. The chest was closed with the usual provision for closed drainage. Re-expansion of the lung again occurred promptly but chyle reappeared in the left pleural cavity. This chyle was aspirated at frequent intervals without any effect on its rate of formation. Accordingly, closed drainage of the left pleural cavity was established, and within seven days there was no further leakage of chyle. She was discharged from the hospital, and for three months got along well. She then developed a severe and extensive dermatitis, which failed to respond to treatment at home so that she was readmitted to the hospital. The last roentgenogram of the chest had shown a shadow in the region of the fissure between the middle and lower lobes and it was thought that she might have some fluid in connection with the disease causing the dermatitis. Two days after entering the hospital she developed jaundice and died within twelve hours of a fulminating hepatitis. At autopsy a prolonged search failed to reveal any trace of a thoracic duct. There was a small amount of chyle in each pleural cavity and in the pericardium. An extensive flat growth of tissue was found in the retroperitoneal space and in the posterior mediastinum. This proved to be lymphosarcoma and was found to involve the lungs, the liver, and many other parts of the body. It was considered to be the cause of the chylothorax. The hepatitis was thought to be due to the large quantity of plasma which she had received four months previously (Figs. 4, 5, 6, and 7).

A third case of chylothorax was seen the next year. This patient was 54 years of age. He was known to have an extensive lymphosarcoma involving the mediastinum and apparently causing the chylothorax. At first, he was treated by repeated aspirations of the chyle but these were of no avail and closed suction drainage was then established. The lung re-expanded and the chyle stopped accumulating. He continued the downhill course and died as a result of the lymphosarcoma.

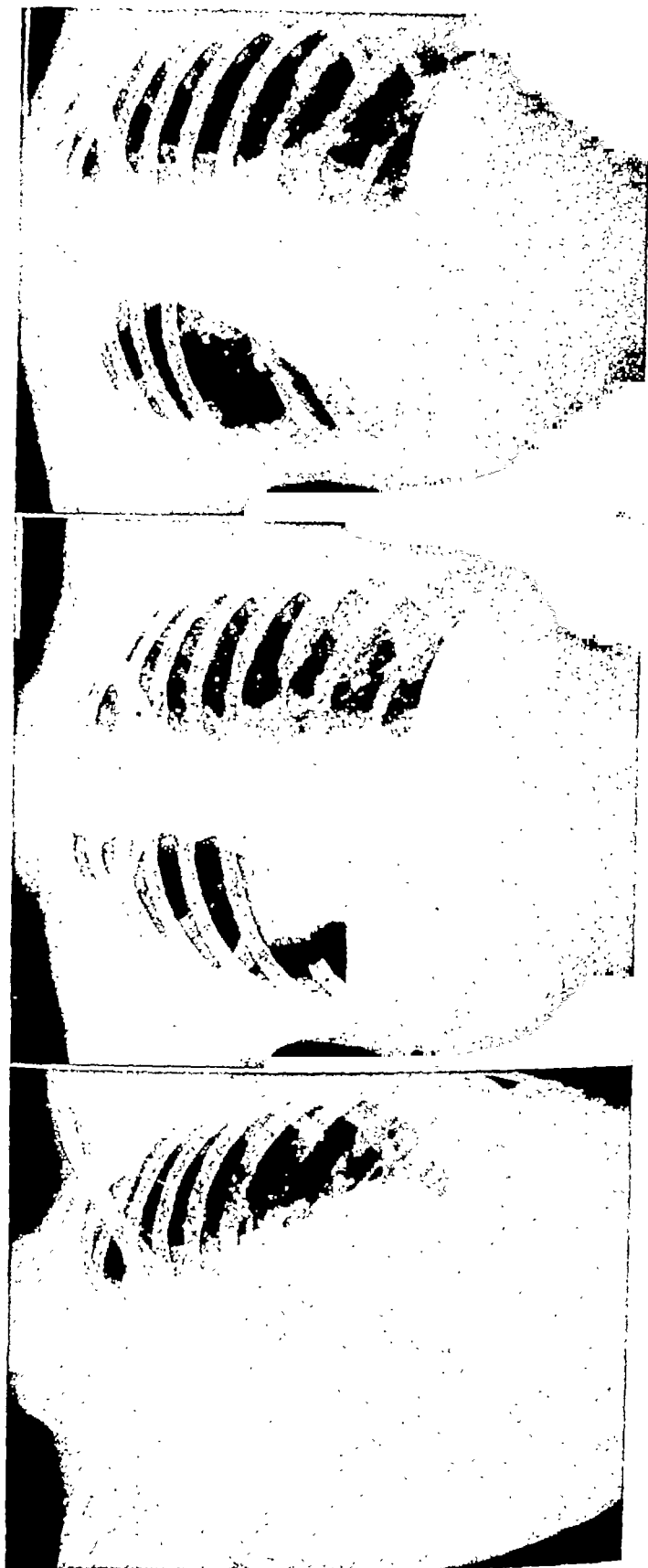


Fig. 1.

Fig. 1 (Case 1).—Chest roentgenogram taken on admission to the hospital showing massive left chylothorax.

Fig. 2 (Case 1).—Chest roentgenogram taken after almost complete replacement of the chyle by air shows marked collapse of the left lung but no evidence of other abnormality.

Fig. 3 (Case 1).—Chest roentgenogram taken eleven weeks after the ligation of the thoracic duct and the lung fully re-expanded except for the extreme apex.

Fig. 2.

Fig. 3.

infiltration of the root of the lung with the chyle made it impossible to secure full re-expansion of the lung at the time of the operation. Full re-expansion of the lower two-thirds of the lung occurred in a few days after the operation with the aid of closed drainage. The apex did not fully re-expand for another two months. This caused no symptoms and she was discharged from the hospital three weeks after the operation. The importance of keeping the lung expanded by frequent aspiration of the chyle is obvious, but, as in our case, it may be impossible. The failure to keep the lung expanded is further reason for speed in stopping the leak of the chyle.

Death from chylothorax, unless the condition is unrecognized and allowed to cause asphyxia, is due to the progressive loss of the fluid, protein, and fat. As chyle is bacteriostatic, we know of no instances in which infection of the chyle has caused death. Before it was demonstrated that the escape of the chyle could be stopped by operation or by closed drainage of the pleural cavity, the importance of replacement of the protein and fat lost in the chyle was great. It is still an important problem. The idea of returning the chyle to the blood stream after its aspiration from the pleural cavity was an obvious one since it is a fluid that normally flows into the blood stream, and it apparently undergoes little change while standing in the pleural cavity. Since this procedure was first reported by Oeken,⁷ in 1908, it has been used in a number of instances with varying results. Its use without mishap by Little, Harrison, and Blalock,⁸ in which they returned 22 liters of chyle to a patient, was reported in 1942. Others have reported the same results but there have been reports of sudden death following this procedure in two patients. The one reported by Whitecomb and Scoville¹¹ came on suddenly after the slow infusion of a small amount of refrigerated, citrated chyle. It was anaphylactic in character. In the other case, reported by Peet and Campbell,⁹ death was sudden in occurrence and no cause for it could be found. It was suggested by Whitecomb and Scoville¹¹ that the patients who are to receive infusions of chyle should first be skin tested with the chyle to determine a possible sensitivity. This seems unnecessary, as one could hardly be sensitive to a natural body fluid which under normal conditions is constantly flowing into the blood stream. These authors also suggest that the fat droplets in the chyle be measured, and if found to be of greater diameter than 3 micra, the chyle should not be used. If coalescence of the droplets can occur in either the pleural cavity, or outside, this would seem to be a wise precaution. In view of the successful, repeated intravenous infusions of freshly aspirated chyle in the case reported by Little, Harrison, and Blalock,⁸ this method would seem to be the best and the least likely to give rise to trouble. However, in some cases there will be precipitation of fibrin, and the fibrin may form large masses. The injection of such fluid into the blood stream could certainly cause disturbances. Therefore, it would seem preferable to administer the chyle after filtration, but as soon as possible after aspiration. It is our belief that, until further experimental work has been done on the causes of reaction following the infusion of chyle, it should not be used.

when she fell down the steps. The delay in the appearance of symptoms could be explained as in the first case, only that in this one there was no extensive infiltration of the mediastinum, and no outlet through the pleura was seen.

In the third patient there was no operation and no autopsy. The patient was known to have an extensive lymphosarcoma involving the mediastinum. The clinical and roentgenographic findings were characteristic and these had been substantiated by biopsy of a cervical lymph gland. It has frequently been mentioned in the literature that chylothorax develops as a result of blockage of the thoracic duct by tumor or by enlarged lymph glands. That such blockage can produce chylothorax seems unlikely in view of the experimental work of Blalock and associates,² who had no success in producing chylothorax in dogs by blocking all available thoracic ducts. It would seem that it would be necessary for the tumor actually to invade the duct, or to fix it so that it could later be ruptured by some sudden movement of the spine, or by sudden increase in intraductal pressure.

Experimentally and clinically, it has been noted that following the rupture or ligation of the thoracic duct there is a striking drop in number, or complete disappearance, of the lymphocytes and eosinophils in the circulating blood. As the lymphocytes are known to be carried through the thoracic duct to the blood stream, it is not remarkable that there should be an effect on their numbers by disturbances of the duct. Why the eosinophils are effected is not known, but their disappearance is of little diagnostic importance since they are usually present in such small numbers that their absence would not be noted. The marked drop in the lymphocyte count can be of great diagnostic value, especially in accident cases when there is a chest injury but no evidence of injury to the thoracic duct. If there is need for thoracic exploration in such a case, and the lymphocytes are markedly decreased in number, a search should be made for free chyle, and the thoracic duct ligated if chyle is found. In our first case the lymphocytes had dropped from 3,744 per c.mm. on admission to the hospital, to 540 six days before operation. Four days after operation there were no lymphocytes in the blood. Eight days later there were 386 per c.mm. and just before her discharge from the hospital eleven days later, there were 1,443. Within the next month the blood picture had returned to normal. In the other two patients the lymphocytes were at a low level during the time the chylothorax was present, but the changes were not as striking as in the case in which the duct was tied.

The local effect of chyle on the pleura is of importance when the leakage is not promptly stopped. It causes an inflammatory reaction which results in thickening of the pleura with a loss in its elasticity and the deposition on its surface of a layer of exudate. Fibrin may be precipitated in masses of varying size and add to the thickness of the coating on the lung. In our first patient, who was not operated on until nine weeks after the discovery of her chylothorax, the lung was collapsed and could not be expanded at all until a tedious decortication had been completed. There was no well-defined "peel" as found in chronic hemothorax or in most cases of chronic empyema. It was necessary to remove a considerable amount of the true visceral pleura. The

longer than two weeks should be allowed for this second stage of treatment since the danger of encapsulation of the collapsed lung is real. When operation is decided upon we feel that the object should be simple ligation of the duct at its lowest accessible point, with resection of a small segment for histologic verification of the structure, and for determination of any pathologic processes in the duct. There is no evidence, of which we are aware, that simple

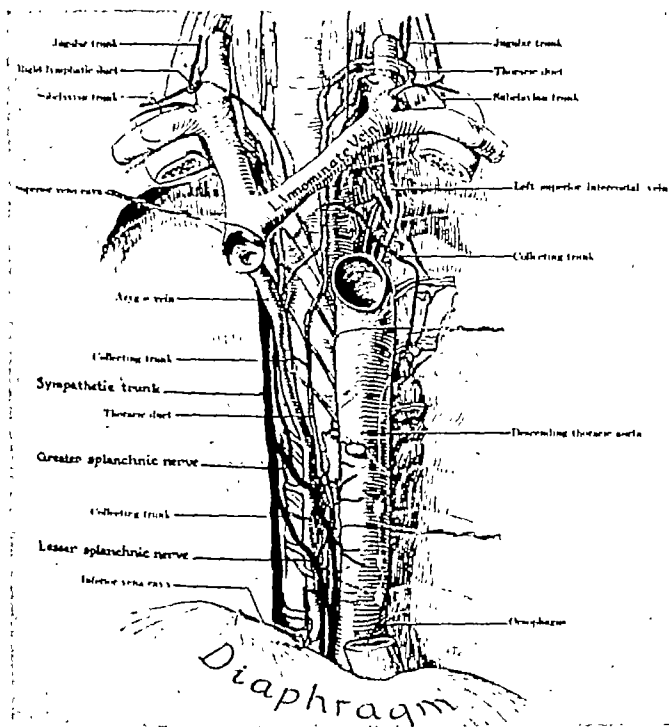


FIG. 8.—Typical course of the thoracic duct in the chest. The close proximity of the right splanchnic nerves to the duct is well shown. (Modified from FIG. 819, in "An Atlas of Anatomy" by J. O. Bolleru Grant, Baltimore, 1912, Williams & Wilkins Company.)

ligation of the duct is not sufficient to stop permanently the flow of chyle. The leakage of chyle from the duct after the closure of its divided ends by silver clips, reported by Whitcomb and Seoville,¹¹ is no condemnation of ligation of the duct, but only of this method of closure. Hodge and Bridges⁴ have recently advocated the anastomosis of the severed thoracic duct and the azygos vein, because they feel that simple ligation may be followed by recanalization of the duct, and further leakage. We do not believe there is

It seems much wiser to prevent depletion than to combat it. A high protein diet and intravenous infusions of amino acids, and an occasional blood transfusion may be used temporarily. Plasma should not be used. It may, as it did in our second case, produce a fatal hepatitis. To prevent malnutrition and to forestall a thickened pleura and permanent collapse of the lung, closed suction drainage or ligation of the thoracic duct should be undertaken early if aspiration is ineffectual.

The thoracic duct is not always the single, well-defined structure that is pictured in standard anatomic works. This was forcibly drawn to our attention during the course of the two operations on our second patient. It always enters the chest through the aortic hiatus of the diaphragm and almost invariably ascends between the azygos vein and the aorta, on the anterior surface of the vertebrae. However, there are variations. Stuart,⁹ in 1907, reported that in his dissections the variations were more frequent than the supposed normal arrangement and that the statement that the thoracic duct was a single channel throughout the chest was misleading. Davis,³ in 1915, described nine types of thoracic duct found in dissecting twenty-two cadavers. However, more recent studies do not confirm these earlier ones. Baldrige and Lewis,¹ in a study of twenty bodies, found a single thoracic duct in each. Van Pernis,¹⁰ in careful studies of 1,081 bodies at autopsy, found a great uniformity except in the small branches of the upper portion. One of us (C. W. M.), working in the department of anatomy at Northwestern University Medical School, studied the thoracic duct arrangement in 135 cadavers. In 78 per cent there was a single duct. In 17 per cent there was a short, single duct which divided into two, which united either below or above the diaphragm. In 5 per cent there was a single duct which divided into two and which ran through the chest. From all of these studies it is apparent that the chance of encountering real difficulty in finding the thoracic duct in the lowest part of the chest should be extremely rare (Figs. 8 and 9).

Having considered the various factors involved in chylothorax, a definite plan for the management of such cases can now be outlined. As it is known that one-half of the recorded cases of chylothorax have recovered after simple aspiration of the chyle, with or without replacement of the chyle, it would be wrong to advocate a more radical measure as the first form of treatment. Except in those cases in which chest exploration must be done for the control of an associated lesion, the immediate ligation of the thoracic duct should not be considered. First, aspiration of the chest should be used at frequent intervals. This should be continued if there is evidence of decrease in the amount of chyle leakage. If not, closed drainage should be started within ten days to two weeks. Whether or not there should be definite suction applied would seem to depend on the ease with which the fluid is removed. In our first case, in which closed drainage was used, there was no suction. In the second one there was, not because of any special indication, but because the operator considered this to be the quicker method of accomplishing the desired result. If suction drainage fails to bring about re-expansion of the lung, and cessation of the chyle leakage, then operation must be done. No

to breathe freely. The patient's nutrition was going down very fast. For the preceding reasons an exploration was done, as has been shown by Dr. Meade, through the right side of the chest. We identified the structures in the region, namely, the axillary vein, the sympathetics, and the aorta, expecting to find some difficulty in identifying the thoracic duct. However, in the region where the thoracic duct should be there was a structure about as big as my index finger. In following this structure we found that it thinned down to the size of the normal thoracic duct, just below the hilum. I sent a specimen for frozen section. It was reported as being inflammatory. The report was erroneous, however, because it subsequently proved to be Hodgkins' disease. However, on the basis that it was inflammatory, I resected the thoracic duct and opened the diaphragm and resected part of the cisterna chyli. There was some leaking of the lymph vessels, the collaterals were picked up and ligated as well as possible, then some Gelfoam was placed where the duct had been resected. The chest was drained, by under water seal drainage, there was very little chyle from the right side of the chest. The left side had to be aspirated twice, then the chyle stopped forming. The patient's nutrition was very poor for the first ten days but, following that, he picked up and gained in weight and is feeling very well at the present time. It is about three and one-half months since the operation.

This case it seems to me brings out two points; the thoracic duct can be resected successfully in the right lower chest. In bilateral chylothorax, if the approach is made on the right side, ligating the thoracic duct at its origin will stop formation of chyle on the left side.

DR. WILLIAM R. RUMEL, Salt Lake City.—I would like to present another case of traumatic chylothorax which was treated successfully by surgery. In evaluating the results of surgical treatment it is very important to differentiate between cases of true traumatic chylothorax and the cases mentioned by Dr. Harper and Dr. Meade, where there is other serious associated disease. It seems to me that the problem in the two types is entirely different.

This case concerned a 5-year-old white boy who was playing with a pocket knife on July 9, 1947. He fell forward in such a manner that the $2\frac{3}{4}$ inch blade entered the base of the neck just above the inner end of the left clavicle approximately between the two heads of the sternocleidomastoid muscle. There was very little bleeding and after the mother applied an antiseptic dressing he continued to play. For four days he was asymptomatic but at that time developed a dry hacking cough and dyspnea. The family physician found evidence of a left hydrothorax. The symptoms were relieved by thoracentesis for another five days. At this time thoracentesis had to be repeated, with removal of 500 c.c. of milky fluid. Because of the appearance of the fluid he suspected the possibility of chylothorax and called me on the phone. I suggested that he send the child in for further treatment, little realizing that I was to find out in the next two or three days that the surgery which I had in mind was associated with 100 per cent mortality, according to the literature. Had I been a little better informed on the subject, I probably would have sent him to my nearest competitor. At any rate, he arrived at the hospital. He was moderately ill, with a temperature of 102° F., a pulse of about 160, and a respiratory rate of 40 with a moderate degree of dyspnea. Thoracentesis again revealed milky fluid which, on subsequent examination, was proved to be chyle.

He was placed on the generally recommended medical therapeutic regime for a period of twelve days during which time the condition deteriorated progressively; so that it was apparent that something further would have to be done. Operation was decided upon, although with a great deal of fear and trepidation, because during that time I was still unable to find a record of a single case that had been treated successfully by surgery. Somehow I overlooked the report of Dr. Ralph H. Loe from Seattle, who had reported a similar case a few months previously. On July 31, 1947, 22 days after the injury, and about one and one-half hours after administration of milk and cream, he was anesthetized and an approach made through the left supraclavicular area. I was delighted on finding the duct without difficulty, including some of its ramifications, and was able to follow it down for a distance of about two inches below the suprasternal notch. At this point it disappeared into a mass of scar tissue. It was possible to dissect down in front of and behind this tissue and while it

10. Van Pernis, Paul A.: Personal communication.
11. Whitecomb, B. B., and Scoville, W. B.: Postoperative Chylothorax, *Arch. Surg.* 45: 747, 1912.
12. Zesns, D. G.: Die nicht operative enstanden Verletzungen des Ductus Thoracicus, *Deutsche Ztschr. f. Chir.* 115: 49, 1912.

DISCUSSION

DR. J. GORDON SCANNELL, Boston.—A recent case of intractable chylothorax which we had at Massachusetts General Hospital may be apropos of Dr. Meade's excellent review of this subject. Unfortunately, our combined efforts were not attended by the same success which he has had.

The patient was a 26-year-old man whose initial complaint was elephantiasis, but who, shortly after admission, was shown to have a right chylothorax and a chylous pericardial effusion. The former failed to subside with repeated aspirations. There was no history of trauma, nor were we able to establish a diagnosis of either tuberculosis or tumor. It was of great interest, however, that his half-brother had died at the same age, 26 years, of a chylous ascites associated with elephantiasis with dependent edema. In the course of hospitalization, massive amounts of chyle were removed from the chest without improvement (slides). Exploration was therefore advised and carried out. At the time of this first exploration his condition on the table was very poor, but the operator, Dr. Souther, was able to ligate the thoracic duct at about the level of the ninth dorsal vertebrae as well as drain the pericardium into the right pleural cavity. There was no evidence of a primary cause of the disease and his condition on the table precluded further search. Immediately following this procedure, he did remarkably well so far as general cardiovascular state was concerned; but the accumulation of chyle continued, in fact rather increased, now in both pleural cavities. In the course of the next six weeks he became a serious nutritional problem—as Dr. Meade has indicated is likely to happen—and we felt that another attempt to stop the leak, without knowing where the leak was, was in order. Our thought was that possibly we could prevent the flow of chyle by resection of the duct or the cisterna chyli below the diaphragm with obliteration of the tract by suture. Therefore, he was re-explored through the right chest and an operative sketch of the procedure is shown (slide). In this case the right tenth rib was resected; the lower thoracic duct was picked up lying between the aorta and the azygos vein and followed down through the diaphragm by dividing the right crus of the diaphragm completely. The upper one-half of the cisterna was resected, all entering branches ligated, and the bed of the duct obliterated. For a matter of two weeks the patient accumulated very little chyle in the chest, but then reverted to the former condition. He subsequently died about six weeks later. At autopsy he had a queer type of fibrosis involving the lymphatics of the lower extremities and a generalized fibrosis of the retroperitoneal and retropleural spaces. In view of the generalized condition of the lymphatic system, it is not surprising that the specific surgical attack on one of the collecting systems failed to arrest the course of the disease.

As an addendum, I might mention that in studying this patient, it was Dr. Churchill's suggestion that we might try to visualize the thoracic duct in the animal and possibly in the patient with radiopaque dye. This was done in a dog. The animal was explored through a left lumbar incision and a small polyethylene tube was put in the cisterna chyli which was distended by a previously eaten fat meal. The end of the tube was brought out to a little capsule beneath the skin. The following day 10 c.c. of 35 per cent Diodrast were injected into the capsule and excellent x-ray visualization of the thoracic duct was obtained (slide). This was repeated one week later and showed no gross damage to the duct. Two weeks later we were able to aspirate chyle from this sampling system.

DR. F. R. HARPER, Denver.—I should like to add one case to this splendid report of Dr. Meade's and the other discussors. The patient was a 42-year-old man who had bilateral chylothorax with collection of tremendous amounts of chyle. We carried on with aspiration for about two months, after which time the chyle was forming so rapidly that it was necessary to aspirate as much as 1,400 c.c. on each side every second day in order to allow the patient

thorax if preoperative thoracentesis shows the left lung to be re-expandible. Identification of all of the duct branches would be more certain and safer than attempting to immobilize the aorta to reach the duct from the left thorax.

DR. ARTHUR S. W. TOUROFF, New York.—I would like to add a report of two additional cases of traumatic chylothorax to the cases already reported. I remember very well, as a house officer more than 20 years ago, a patient who had had the left side of the neck explored by one of our senior surgeons. Operation was followed promptly by leakage of milky fluid from the wound. The condition of the patient slowly deteriorated and he died of inanition in a period of approximately four weeks. From that time until World War II, I did not see another case of injury of the thoracic duct. While overseas in 1944, I had occasion to observe a patient who had sustained a perforating wound of the left interscapular region, the site of exit of the missile being in the suprasternal notch. By some miracle, the patient had escaped injury to the trachea and great vessels. Except for the fact that he was intensely dyspneic and cyanotic, he had no complaints. X-ray examination revealed a dense shadow in the left chest; and, at his bedside there was a test-tube filled with fluid which was assumed to be pus. The patient, strangely enough, had no fever. Accordingly, it seemed obvious that he was not suffering from empyema. Furthermore, as the fluid was carefully observed, its chylous nature was obvious.

Because of the severe dyspnea and cyanosis, the left chest was aspirated and approximately 4,000 c.c. of chylous fluid removed. This was followed by prompt disappearance of dyspnea and cyanosis. I did not know whether anyone had ever administered chyle intravenously before; and, remembering the patient of 20 years ago who had died of inanition, felt that we should attempt to give this patient his own chyle by vein. With great trepidation, we started the slow, intravenous administration of chyle which had been removed from the patient's chest. Much to our astonishment and delight no untoward reaction was observed; and, at the end of a few hours, we had gradually opened the clamp more widely until the chyle was flowing into the vein at a rapid rate. Aspiration of the chest was performed daily and, at the end of approximately ten days the lung was completely re-expanded and the patient well. A few months later we had a case that was practically identical in all details.

I assume that in both cases, the injury must have been very close to the point of junction of the thoracic duct with the left subclavian vein, because it was inconceivable that a missile entering the body close to the scapula, and making exit in the suprasternal notch could have injured the thoracic duct at any other point. Fortunately, conservative treatment, consisting of aspiration of the chest and the administration of chyle intravenously, resulted in the recovery of both patients.

DR. MEADE, JR. (closing).—I should like to add one case report from Dr. Robinson, who is associated with Dr. Jones; I had hoped he would be here.

The question of the etiology of chylin and of chylous ascites, in many cases cannot be explained on the basis of anything I know of at the present time. The use of the lipophylic dyes, I think, is a valuable procedure and I think I shall use them the next chance I have, which may not be for many, many years.

was not possible to actually demonstrate a tear in the duct, it was assumed that it communicated with the pleural space at this point. Two silk ligatures were placed around the area, one below the scar tissue and one above. These were tied firmly and the incision was closed. Fortunately he made a very uneventful recovery. It was not necessary to aspirate the chest even on one occasion. Since that time he has developed in a perfectly normal manner.

If we add this case to Dr. Meade's—that is, the one which was not associated with another serious underlying lesion—and include the cases reported by Loc, Peet and Campbell, Lampson, and Baldrige and Lewis, it will make a total of six cases of traumatic chylothorax treated surgically. In each case a successful result was obtained with the exception of that reported by Peet and Campbell where death occurred suddenly following intravenous administration of chyle, presumably due to anaphylaxis. In evaluating the risk of surgery in these cases it would seem proper to eliminate this death inasmuch as it apparently was due to the rather dangerous procedure of parenteral administration of chyle.

From this, it would appear to me that perhaps we could be even a little more aggressive than Dr. Meade suggests, and go right ahead with surgical treatment in these traumatic cases as long as we can show that the mortality rate is below that of the ordinary medical measures, that is, around 40 to 50 per cent, and as long as these patients obtain good results following surgery. It seems that the only objection to this recommendation would be continued acceptance of the statement that has been made repeatedly in the medical literature to the effect that any direct surgical attack on the thoracic duct is associated with 100 per cent mortality. If we look into the basis for this statement it will be found that it is based on several cases treated by various surgical procedures, in only one of which—that of Whitcomb and Seoville—the duct was actually ligated.

DR. R. G. KLEPSEK, Washington, D. C.—I would like to add to this list one additional case of traumatic chylothorax successfully treated by ligation of the duct and also to suggest a method to make the diagnosis and the operation somewhat easier. We have at the present time two patients with chylothorax in the Naval Medical Center Hospital at Bethesda; one is a traumatic case upon whom I operated last Friday, six days ago. He is a 290 pound man who was in an auto accident in July, 1948. He had been treated as a cardiac case because of retrosternal pain for six months before fluoroscopy was done. A diagnosis of chylothorax was made after aspiration of the fluid. The fluid was repeatedly aspirated, about 1,000 c.c. being removed every other day; the chyle continued to leak into the right chest. During this time we came upon a method of diagnosis by feeding these patients fat which contained lipophilic coal-tar dyes. This is not new; Fletcher in England had given Sudan III to a patient who had chyle in the urine, and upon recovering the Sudan III in the urine made the diagnosis of chylouria in 1930.*

When these dyes, using Sudan III or other colored dyes, are ingested, the chyle, about one to two hours later, becomes colored. Only the fat is colored and by churning the chyle the fat pellicle can be removed; it can be colored red or green or blue or any color desired. The vegetable dyes are valueless. Oleomargarine dyes were used and were not reobtained in the chyle; the coal tar dyes, of which Sudan III is one, are the best. We used a green dye before operating, putting it in olive oil and giving it through a duodenal tube several hours prior to operation. When the chest is opened the ducts and the chyle are stained, making identification of the ducts much easier. The dye remains attached to the fat, and no other structures are stained. Because of contrast, a green dye seems preferable.

We ligated a large duct and three smaller branches. There was no cortex or peel over the lung in spite of the fact that this lung had been down from six to nine months before, and we had no trouble re-expanding the lung. The chest was closed without drainage. A roentgenogram was taken yesterday and shows no fluid in the chest. In this case the chylothorax was on the right side. Because the lower portion of the duct is so much more accessible, I believe the right-sided approach is preferable for duct ligation in a left chylo-

*Fletcher, H. Morley: Proc. Roy. Soc. Med., 1930.

that some type of tumor was diagnosed preoperatively, and after the pathologist had established the correct diagnosis of tuberculous lymphadenitis, the surgeon and the radiologist acknowledge their mistake by calling it a tuberculoma.

Tuberculomas have been reported in every organ of the body except those which have natural tissue immunity to invasion by tubercle bacilli, for example, the pancreas.

There is an implication in the term "pulmonary tuberculoma" that it is a solitary lesion. The chest roentgenograms give such an impression, but after the lobe, segment, or wedge containing the tuberculoma is removed and examined grossly, smaller similar fibrocaseous nodules will be found in 60 per cent of the specimens as satellites close to or at some distance from the main nodule (Figs. 1 and 7). So "tuberculoma" is a type of fibrocaseonodose tuberculosis in which one nodule is outstanding, attracting the attention of the roentgenologist by its size, its sharp outlines, and the lack of surrounding parenchymal infiltration. It may be found in any portion of either lung; its location in the 48 cases in this series was as follows: right lung 29, with 13 in the upper lobe, 3 in the middle lobe, and 13 in the lower lobe; left lung 19, 13 in the upper lobe, 5 in the lower, and 1 interlobar.

The Ghon nodule, which is the encapsulated primary focus, is rarely a surgical problem. But the increasing occurrence of primary infection in adults, plus the tendency in this age group to delayed retrogression and calcification of the nodule, will bring the primary round lesion to the attention of the radiologist and the chest surgeon.

DEVELOPMENT OF PULMONARY TUBERCULOMAS

It is generally accepted that the pulmonary tuberculoma, excluding the Ghon nodule, is a reinfection lesion acquired by the aerogenous route. It is the structure of the lung with its air spaces and its pleural covering that sets off the tuberculoma of the lung as different in its initial phases from tuberculoma of other organs. This initial phase, whether a primary infection or a reinfection, is a caseous pneumonia of varying extent with tubercle formation somewhat later. The original pneumonia, if successfully handled by the body, clears up, leaving a few visible scars. But the body may be only partially successful in limiting the process by gradually walling in the area of pneumonia by fibrous encapsulation. When followed in serial roentgenograms this walling-in appears as an irregular shadow which gradually rounds out, becomes denser, more homogeneous, and sharper in definition, with the appearance on roentgenograms of that of a discrete round homogeneous density in otherwise fairly normal parenchyma. This is the tuberculoma stage, representing a truce in the struggle between the body and the tubercle bacillus, with the residual bacilli secure behind their fibrous wall. In streptomycin treated cases, assays of these nodules for the drug were negative. The nodule may remain apparently unchanged for years, lulling the doctor into false ideas of its stability and innocence. But in many of the lesions, considered an immature type, there is microscopic evidence that they are sluggishly active; caseation is continuing on the inner side of the fibrous wall, while on the outer side more fibrous tissue

THE SURGICAL TREATMENT OF ROUND TUBERCULOUS PULMONARY LESIONS (TUBERCULOMAS)

COLONEL HUGH W. MAHON (BY INVITATION), AND COLONEL JAMES H. FORSEE
MEDICAL CORPS, DENVER, COLO.

THE TERM TUBERCULOMA

TUBERCULOMA,⁹⁻¹¹ round pulmonary focus,³ solitary focus of tuberculosis,¹² calcified pulmonary abscess,^{5a} Assman focus,¹ Simon focus,¹³ and the Ghon nodule⁶ are similar in that they all represent the focalization of a tuberculous pneumonia by fibrous encapsulation, appearing as a rounded subpleural pseudotumor located in some portion of the lung. The Assman focus is infraclavicular, the Simon focus is apical, and the Ghon nodule is located usually in the mid-lung field. The term "cold abscess" is frequently applied to similar nodules in solid organs and tissues; it corresponds to the "unripe" phase of pulmonary tuberculomas.

There is some objection to the use of the term tuberculoma. Dorland⁴ defined it as a "neoplasm caused by the bacillus of tuberculosis." But this is too simple and half wrong; it is not a neoplasm, although it is frequently diagnosed as such in the x-ray interpretation of rounded parenchymal densities in the lungs (Holmes and Robbins¹⁰).

These tuberculous nodules are the result of a chronic inflammatory process of granulomatous type, which follows a characteristic pattern, associated with, but not the exclusive property of, an infection caused by the *Mycobacterium tuberculosis*, for example, infection with *Coccidioides immitis* can produce a nodule identical with those due to tuberculous infection. Webster's dictionary¹⁴ defined tuberculoma as a "localized growth of tissue caused by the bacillus of tuberculosis; a tuberculous nodule." But this would include microscopic submiliary tubercles as well as the largest of the tuberculous nodules, which in this series was 6.0 by 4.5 cm. Obviously a nodule to be considered a tuberculoma must be of a size sufficient to be recognized in roentgenograms, so should be at least 0.3 cm. in diameter to attract attention to it. In this series of 48 tuberculomas the size varied from 1.0 cm. to 6.0 by 4.5 cm.; the average size was 2.1 cm.

Recently reports of mediastinal tuberculomas have appeared in the literature. Blades and Dugan² and Grace⁷ reported such cases; Blades's case was apparently a hyperplastic reactive tuberculous lymphadenitis and Grace's case a fibrocaseous lymphadenitis. It is not usual to consider an enlarged tuberculous node or a mass formed by conjoined tuberculous nodes as a tuberculoma; otherwise tuberculomas of the neck, axilla, etc., would be a common diagnosis. Apparently the term tuberculoma as applied to the mediastinal nodules implies

From the Laboratory and Surgical Services, Fitzsimons General Hospital, United States Army.

Read at the Twenty-ninth Annual Meeting of The American Association for Thoracic Surgery, New Orleans, La., March 29, 31, 1949.

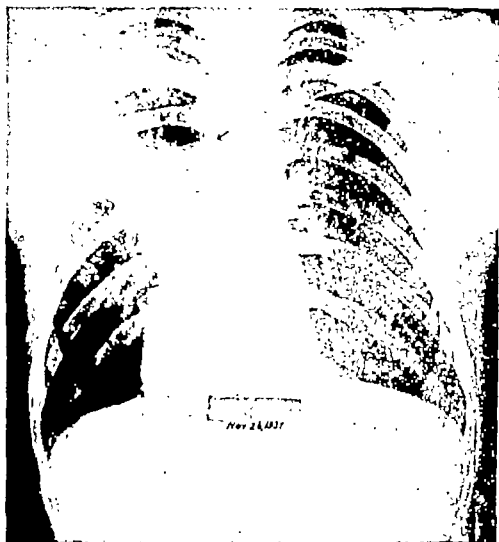


Fig. 2 (Case 1).—Considerable infiltration, left upper lobe. Sputum positive. (See Fig. 4.)

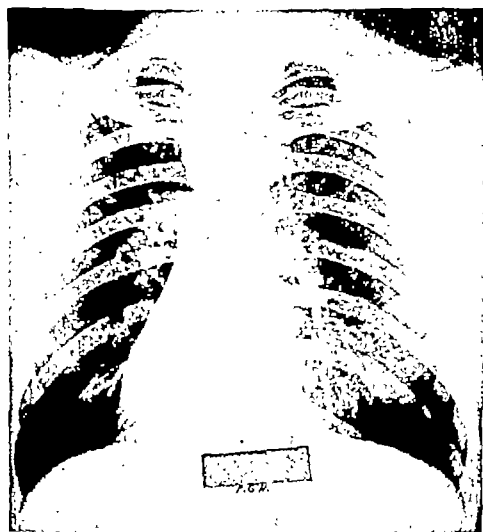


Fig. 3 (Case 1).—Residual rounded density from a tuberculous caseous bronchopneumonia. Lobectomy performed. Caseous tissue of nodule positive for acid-fast bacilli. (See Fig. 2.)

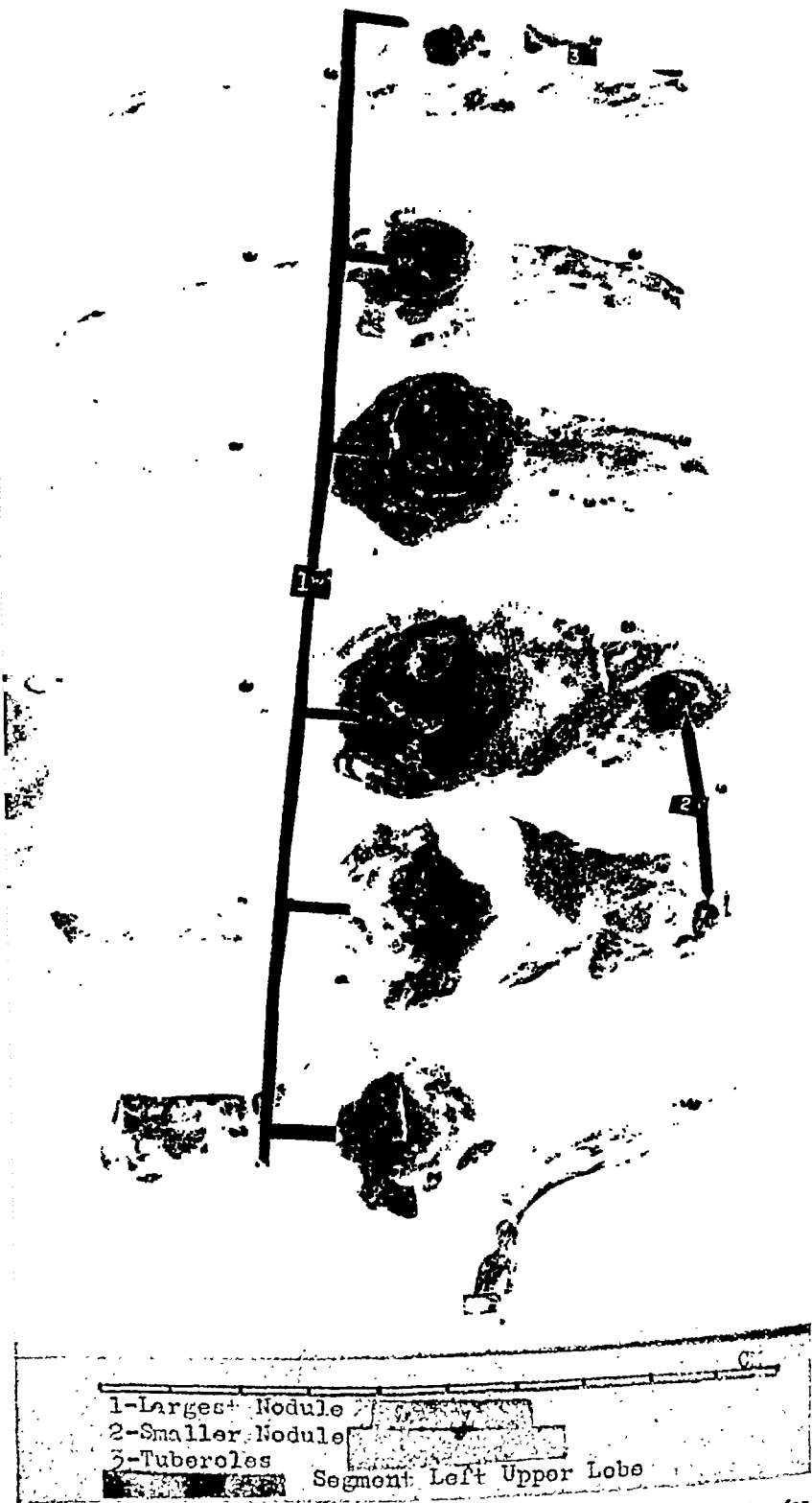


Fig. 1.—Tuberculoma with distant satellite nodules. Material from lesion positive for acid-fast bacilli.

pneumonia, with each small unit encapsulating and fusing with its neighbor to produce a rounded nodule of approximately the same size as the originally involved focus.

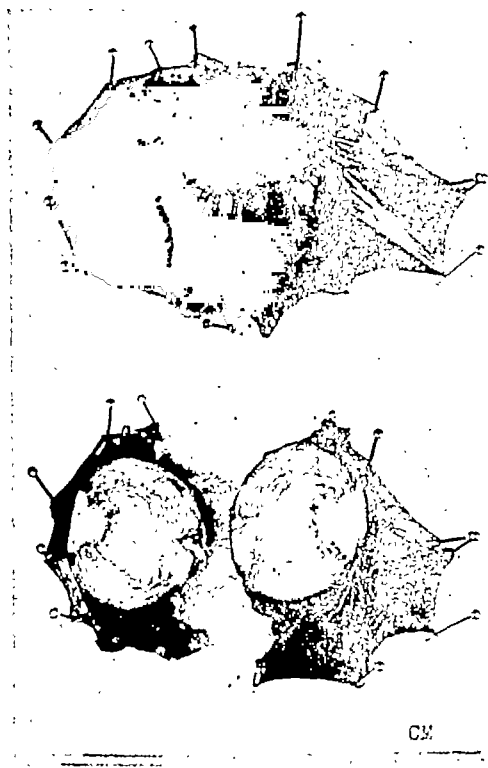


FIG. 5.—"Mature" tuberculoma. Segment of left lower lobe. (See FIG. 4.)

3. It begins as an extensive area of tuberculous bronchopneumonia which proceeds to cavitation. In one-half of such cases it was in the stage of cavitation when first detected by a routine x-ray examination. Occasionally spontaneously, but usually as a result of compression therapy—pneumothorax, pneumoperitoneum, or thoracoplasty—the bronchus which drained the cavity became blocked, and the cavity shrunk, its caseous contents were retained and were concentrated, and gradually the lucency of the cavity lumen was blacked out. The result is a round focus not recognizably different at this stage from the similar rounded foci of 1 and 2, mentioned previously. At the same time the positive sputum was converted to negative.

is being added, but the balance in the centrifugal enlargement is so evenly maintained that neither change in size nor alteration in density may be detectable by x-ray examination.

Of the 48 cases of tuberculous nodules reported in this paper, 60 per cent were well-developed round lesions when first observed. There were no available roentgenograms or too infrequent roentgenograms to follow the development of these lesions. The remaining 40 per cent were followed by serial roentgenograms for several years. Observation of the x-ray examinations in this latter group, plus the microscopic examination of the nodules after they were removed, showed three methods of development:

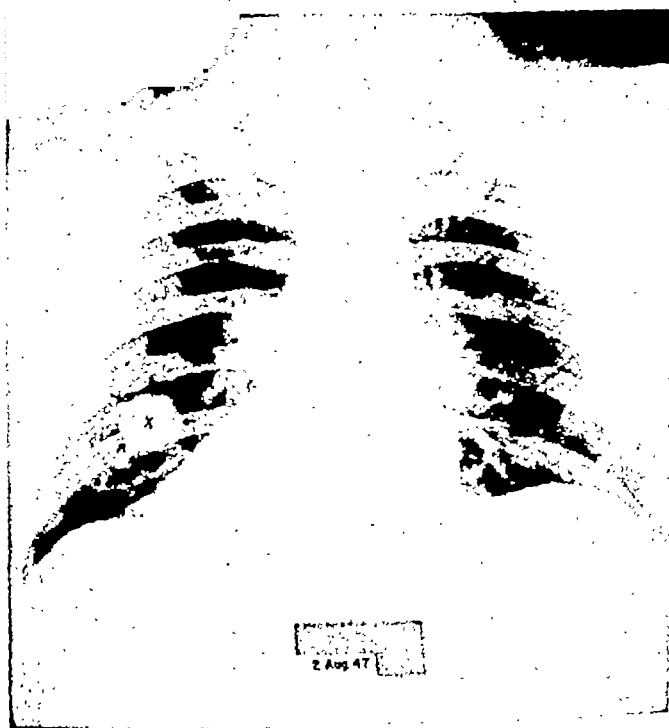


Fig. 4.—Round lesion known to be present and slowly increasing in size for six years. Material from nodule negative for acid-fast bacilli. (See Fig. 5.)

1. Originally there was a rather extensive tuberculous caseous bronchopneumonia with positive sputa and accompanied by the usual symptoms of an active tuberculous infection. Under the usual bed rest regimen, the rather extensive pulmonary infiltration slowly cleared leaving a rounded density much smaller than the original, involved area (Case 1, Figs. 2 and 3).

2. In a clear lung field an indistinct, faint, irregular, hazy spot or a cluster of small (several millimeters) densities appeared. Symptoms were as a rule absent or there was minimal malaise, slight nonproductive cough, occasional 99° F. temperature, and negative sputa, and occasionally positive cultures were obtained from gastric washings. This fuzzy area enlarges and increases in density, gradually rounding out until it finally becomes a sharply circumscribed round shadow. Microscopically the process is a caseous pneumonia but smaller pulmonary units are involved, designated as a lobular or bronchiolar

contents there is a narrow zone of epithelioid cells and occasional typical Langhans giant cells, but usually these giant cells appear shrunken and distorted. In places this intermediate epithelioid cell layer is replaced by a narrow zone of granulation tissue. The caseous center tends to inspissate and calcium phosphate is deposited in it. This calcium deposition may be irregularly scattered through the caseous center or be deposited in concentric rings (Liesegang's rings). The calcification in our cases was never as general, complete, and dense as in a Ghon nodule; rarely was decalcification required before the sections could be cut. This targetlike, partially calcified nodule is the "ripe" or mature tuberculoma and smears and cultures of these lesions were always negative for tubercle bacilli (Figs. 4 and 5). The ringlike calcifications may be visible in roentgenograms. Haight and Farris's case⁹ was of this type, as were Cases 1 and 2 of Graham and Singer.⁸ Such calcified fibrotic nodules are mausoleums erected around dead bacilli.

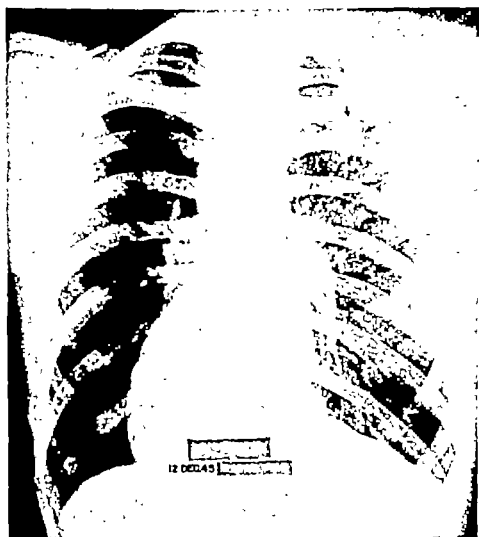


Fig. 8 (Case 3).—Round lesion, right, which cavitated. (See Fig. 9.)

Occasionally the caseous center liquefies uniformly, and the nodule appears as a thin-walled fibrous sac filled with diffuent pale yellow material more like pus than caseous material (Case 2). This type is the most unripe and usually contains innumerable acid-fast bacilli (Figs. 6 and 7). Its removal may be complicated by the spilling of its contents with the possibility of the development of a tuberculous empyema and an infected operative site with pleuro-

FATE OF TUBERCULOMA

When the nodule is formed by the encapsulation of broncho or bronchiolar and alveolar caseous pneumonia, the outlines of the consolidated alveoli, filled with ghost epithelioid cells, can be still identified in the microscopic sections. It may persist in this phase for months. Ultimately the outlines of the previous

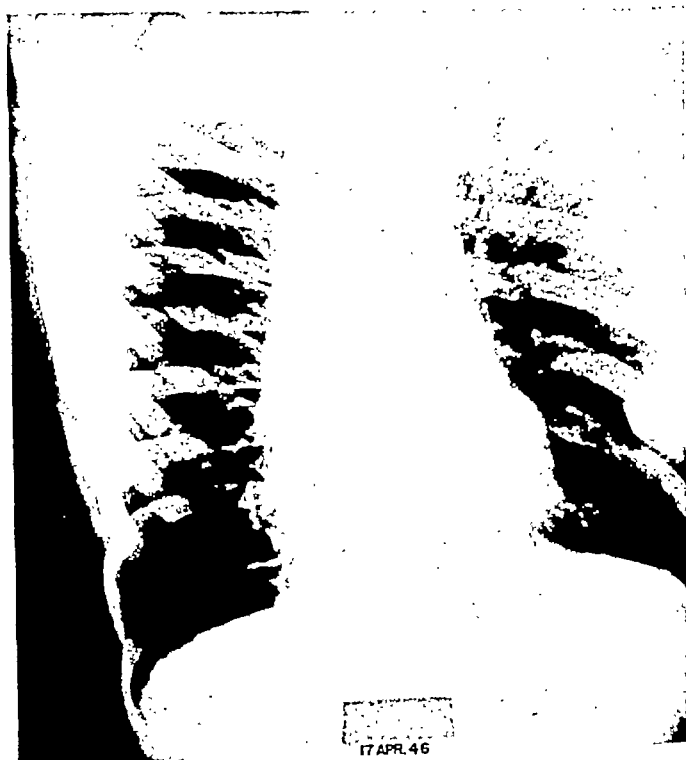


Fig. 6 (Case 2).—Round lesion, right. Asymptomatic. An "unripe" tuberculoma. Material from lesion positive for acid-fast bacilli. (See Fig. 7.)

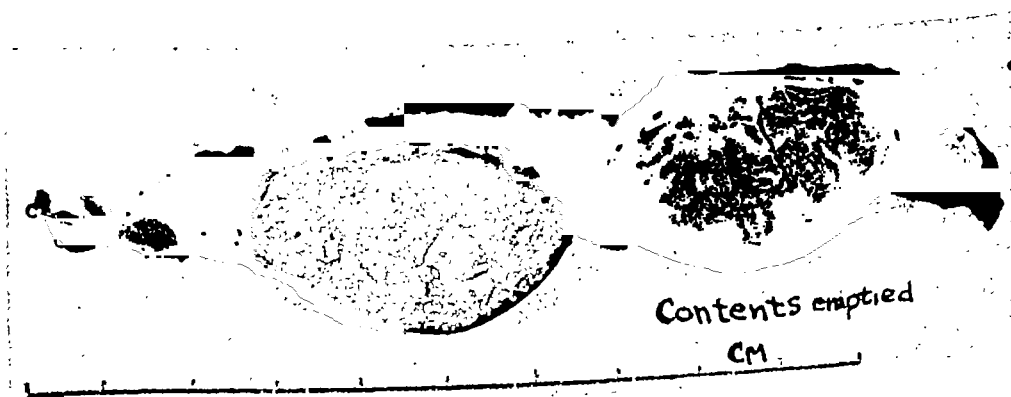


Fig. 7 (Case 2).—"Unripe" tuberculoma. (See Fig. 6.)

alveolar and lobular structure and contained cells disappear and the center of the nodule is uniformly caseous and structureless. The periphery is composed of concentric layers of fibrous tissue with some hyalinization and is infiltrated by plasma cells and lymphocytes. Between the fibrous wall and the caseous

eral "slits" following the curve of the wall of the nodule (Fig. 10). Smears and cultures of these irregularly softened nodules are usually positive for acid-fast bacilli. There are always small patent bronchi or bronchioles in close proximity to the wall of the tuberculoma. Often portions of the wall are quite thin. With liquefaction close to a thin wall, communication can be and is established with the bronchi, with resulting escape of bacilli containing material and reactivation of the tuberculous process.

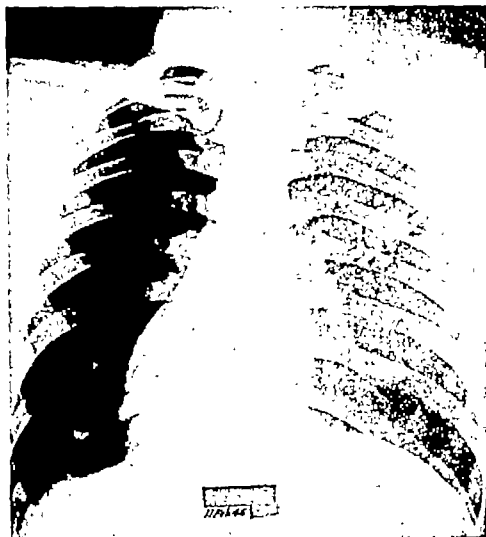


Fig. 10.—Rather rapid enlargement of round lesion, right. Note slitlike space at periphery. Material from center of excised lesion positive on culture for acid-fast bacilli. A "ripened" tuberculoma which has begun to soften and cavitate.

In an occasional slide, submiliary tubercles have been observed in the walls of blood vessels close to the nodule. This infers the possibility of hematogenous spread although no such case occurred in our series.

Tuberculomas originating from encapsulated caseous pneumonia are always close to the pleura in the periphery of the lung. Those that bulge slightly beneath the pleura usually have a white to yellow hyalinized plaque marking the fusion of the wall of the nodule with the overlying reactively thickened pleura. Those that do not quite meet the pleura usually have a dimpled pucker over them with a small white fibrous pleural thickening at the bottom of the dimple. The hyaline plaque often extends as a "V" into the nodule and may be so dense that the nodule is grossly diagnosed as a chondroma (Figs. 4 and 5).

Normal lung tissue surrounds the wall of the nodule. Usually interlobular trabeculae radiating from the wall of the nodule are thickened. Close to the

cutaneous fistula. In the case of Maurer-Mast and Franklin¹¹ this occurred. In Case 3 the liquefaction was incomplete; communication with a bronchus was re-established and a thick-walled cavity remained (Figs. 8 and 9).

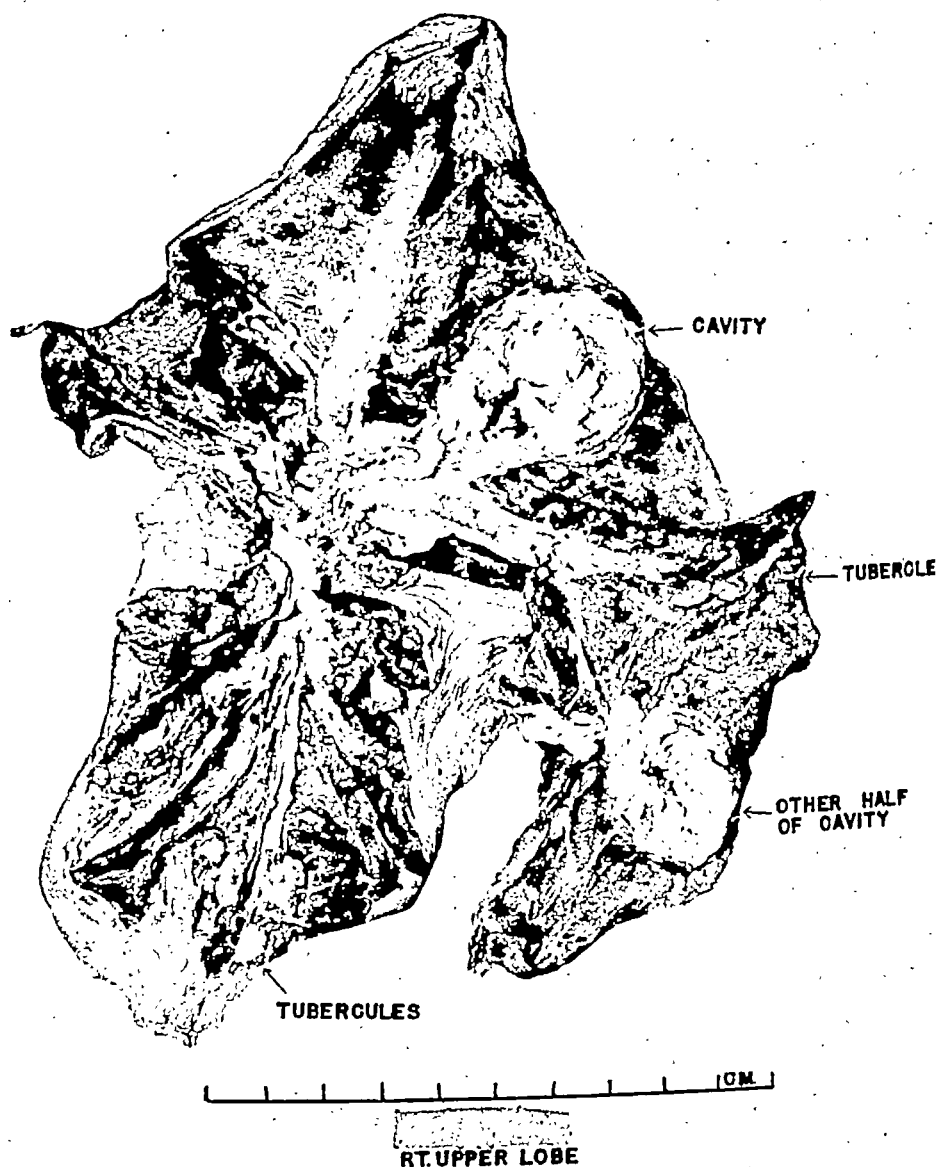


Fig. 9 (Case 3).—Tuberculoma which cavitated and established communication with a bronchus. A tuberculous endobronchial disease is also present. (See Fig. 8.)

Unfortunately the large majority of the solid nodules are slow to calcify and mature; the longest observed in this series was eight years when it began to soften and the sputa became positive. They often show small areas of softening and liquefaction, evidenced in the roentgenograms by small eccentric lucencies which may shift within the nodule when followed by serial roentgenograms. It is rather characteristic that these lucencies are located at the junction of the caseous and fibrous zones and often appear in the x-ray shadows as periph-

6. Skin sensitivity tests are usually not particularly helpful. The tuberculin skin test may be positive; in this group 70 per cent were positive, many only with second strength tuberculin (.005). Histoplasmin and coccidioidin tests may be equally positive. The coccidioidin skin test may be positive and the tuberculin negative, yet the lesion proves to be tuberculosis. In other words, skin tests are as often confusing as they are helpful.

7. Needle biopsy. Although employed by others, it is not used at Fitzsimons General Hospital.

8. Physical examination. It is usually negative. No definite findings as a rule are present except in those cases of a residual nodule following extensive active tuberculosis.

9. Sedimentation test. It is usually normal or only slightly elevated.

DIFFERENTIAL DIAGNOSES

The round lesions found in surgical specimens removed, since 1943, at Fitzsimons were: (1) atelectasis of a nipple of lung parenchyma, adherent to the parietal wall, which had been "strangled" by adhesions; (2) bronchial adenoma not visualized by bronchoscopy; (3) echinococcus cyst; (4) peripheral bronchiogenic carcinoma; (5) peripheral fibrosarcoma with no other primary site established; (6) solitary metastasis of a renal adenocarcinoma; (7) a filled bronchiogenic cyst; (8) a venous aneurysm in the middle of a lower lobe; (9) calcified cast in a cystic bronchiectasis; (10) chondrohamartomas (these are also peripheral subpleural round lesions with suggestion of central cavitation at times); (11) nodular lesions of coccidioidomycosis (their differentiation from tuberculoma depends on the recognition of the spherules in the tissue sections or by culture of *Coccidioides immitis* from the caseous contents); (12) encapsulated interlobar tuberculous pleurisy; (13) pyogenic lung abscess. In spite of employing every possible diagnostic procedure to arrive at a correct diagnosis, each of the aforementioned lesions was suspected at some time during the hospitalization of the patient to be a tuberculoma, and only their examination after removal established the correct diagnosis.

REASONS FOR SURGICAL REMOVAL

1. There is no medical treatment that will clear up these nodules. Streptomycin has no effect on the nodule and from assays on five of these encapsulated lesions, it appears that streptomycin does not penetrate their fibrous wall.

2. They are potentially dangerous. They may persist for years apparently unchanged and then cavitate, break their fibrous barrier with a resulting acute tuberculous pneumonic flare-up.

3. The employment of every available diagnostic procedure may still not establish a correct diagnosis. Only by excision of the lesion and its examination by the pathologist will the specific diagnosis be obtained.

4. Parenchymal round lesions, by the law of averages, are mostly tuberculomas, but they can be neoplasms. Seven of the solid round lesions, removed during the same period as the 48 tuberculomas, were tumors; 4 of these were

wall there is a narrow zone of partially atelectatic alveoli, due to compression by the nodule. The lymphocytes and plasma cell reaction usually extend into the slightly thickened fibrosed walls of these alveoli. It is common to find submiliary epithelioid giant-cell tubercles in the surrounding parenchyma as evidence of continued activity of the tuberculous process. As mentioned previously, satellite fibrocaseous nodules 0.2 to 0.7 cm. in diameter may lie close to the main nodule. Occasionally the satellites fuse with the main nodule, explaining the enlargement of some nodules.

The description of a tuberculoma is entirely individual; each one differs. The general characteristics are: (1) a fibrous wall hyalinized in varying degrees; (2) a caseous center of varying consistency from diffuent to dry, granular, and chalky; (3) calcium deposition of varying degree and arrangement; (4) anthracotic pigmentation depending on the degree of anthracosis in the rest of the lung and on the area of the parenchyma originally involved; (5) hemosiderin pigment deposition, usually scanty, and associated with the presence of granulation tissue in the lining; (6) cavity formation of varying degrees, usually irregular, slitlike, and eccentric, rarely completely cavitated; (7) a pleural cap of hyalinized fibrous thickening over the bulging dome or a dimpled area over the deeper tuberculoma.

DIAGNOSIS

As aids in the diagnosis there are:

1. A careful history. But in the majority of cases no history of personal or familial tuberculosis was obtainable nor could any history of definite contact be found.

2. Roentgenograms. It is always well to remember that there is more tuberculosis present than the roentgenograms indicate. Posteroanterior roentgenograms of the chest establish the presence of the nodule and of a calcified Ghon nodule or a Ranke complex. In one case, calcifications were demonstrated in the spleen. Stereoscopic roentgenograms will help in localization. Oblique, lateral, and apical films may better visualize the nodule and locate it fairly accurately. Potter-Bucky technique and tomograms help in better delineation of the nodule and its internal structure, and in locating the depth of the lesion from the chest wall.

3. Fluoroscopy. It will show that the nodule does not pulsate and that it moves with the lung.

4. Bronchoscopy. Its value is chiefly negative. As these nodules are usually at the periphery of the lung they are beyond the limited field of the bronchoscope. If the nodule has cavitated and communicates with a bronchus, bronchoscopy may determine which bronchus is draining and by obtaining material for culture may establish a diagnosis of tuberculosis. Preoperatively it is used to determine if endobronchial tuberculosis is present.

5. Bronchography. It is usually used to rule out other conditions, as bronchiectasis and bronchial cysts. It is the most accurate way to localize the bronchopulmonary segment involved.

physical check-up, for physical clearance previous to separation from or re-enlistment in the armed service, or during screening on admission to hospital for some other condition.

In known cases of tuberculosis their development was followed by repeated x-ray examinations; they are the end result of a tuberculous pneumonia or of the solidification of a tuberculous cavity during compression therapy.

The 48 specimens illustrate the individuality of the process and the various phases of development from immaturity to "ripeness." The slowness to mature and the tendency to cavitate are emphasized.

The marginal slitlike cavitation is offered as a differential point in the x-ray diagnosis of these lesions and occurred in lesions well on their way to maturity.

The ages varied from 19 to 54 years, average age being 32 years. Forty-three men and 5 women composed the group. This preponderance of men reflects the Army source of the patients. As to races, 45 were white, 1 colored, and 1 Japanese. This bears out the lessened ability of the Negro race to focalize pulmonary tuberculosis.

The potential danger due to the presence of viable bacilli in the majority of the tuberculomas is emphasized (Case 3, Figs. 8 and 9).

The comparative safety of the operative removal of these nodules is reflected by the following facts: no operative deaths, no postoperative tuberculous pleurisy, and no immediate postoperative disseminations.

CASE REPORTS

CASE 1.—This 36-year-old medical officer gave a negative familial history for tuberculosis. In 1930, he had pneumonia with no residuals. About Nov. 8, 1937, a dry hacking cough began with malaise and slight fever. The temperature and cough persisted and he lost five pounds. He was admitted to Walter Reed General Hospital, Nov. 18, 1937. Roentgenographic examination revealed extensive infiltration of the left lung from the apex to the level of the third rib with linear nodose appearance and a suggestion of a cavity. Sputa were repeatedly positive for acid-fast bacilli. He was transferred to Fitzsimons General Hospital on Dec. 20, 1937, and a left pneumothorax was induced Dec. 23, 1937. He gained weight, cough cleared, and sputum became negative by July 18, 1938. He was discharged on Nov. 20, 1939. Smears and cultures for tubercle bacilli were negative. He resumed normal activity and check x-ray examinations showed re-expansion of the lung with some fibrosis in the upper left lobe at the site of previous tuberculosis.

He was readmitted March 11, 1945, because sputa had become positive. Sputa and bronchial aspirations were positive between April and August, 1945. He had increased cough and expectoration and there was an infiltrative process in the left upper lobe, and roentgenogram of the chest on April 4, 1945, showed a circumscribed density in the first left anterior interspace (Figs. 2 and 3). This slightly regressed by Aug. 10, 1945. Sedimentation rate was 4/60. On Sept. 6, 1945, a left upper lobe lobectomy was done.

Pathologic Examination.—Specimen was the left upper lobe weighing 135 Gm. Fibrous adhesions were attached about the apex. A firm 1.0 cm. nodule was located in the upper one-half of the posterolateral segment. It had a fibrous wall enclosing grayish yellow caseous material. Smears were positive for acid-fast bacilli. Calcium was present and cholesterol crystals. A bronchiole lay against the fibrous wall. Occasional satellite tubercles were present in adjacent parenchyma. About this nodule in the apical segment, there were scattered fibroed tubercles and an occasional conglomerate fibrocaseous tubercle.

malignant (1 sarcoma, 2 peripheral bronchiogenic carcinomas, and 1 metastatic round lesion of a primary renal carcinoma). So of 55 round solid lesions from the periphery of the lung, 4 were malignant, or 7 per cent. Effler, Blades, and Marks⁵ in their series of 24 round lesions found 15 per cent malignant and approximately 30 per cent tuberculomas but their series included bronchiogenic cysts and round lesion located in the paravertebral gutter.

5. The cure of the visible disease is possible as it is confined to an area readily accessible to surgery. Its removal can be accomplished by surgical means which will not cripple the patient and can restore his peace of mind and usefulness.

6. The operative risk is low; it is no greater than that of appendectomy. In this series there were no operative deaths. The patients are usually excellent operative risks.

SURGICAL MANAGEMENT

The management of these cases depends on an appreciation of the underlying pathology.

Fortunately these lesions are peripheral, subpleural, with few or no adhesions to the parietal pleura. Palpation of the lung usually can detect the extent of the main nodule and the presence of satellite or additional nodules. Surgical judgment must decide the amount of tissue to be removed. A wedge excision is sufficient for the mature fibrocalcific tuberculoma. A segmental or total lobectomy may be necessary where the area involved is too extensive for a wedge resection. Enucleation is to be avoided due to the possible presence of microscopic tubercles in the adjacent parenchyma.

In this series wedge resection was employed in 30 cases, segmental resection in 2, and lobectomy in sixteen. In 1 case the tuberculoma was located along the line of an incomplete minor fissure and the removal of the upper and middle lobes was necessary.

The danger of postoperative tuberculous pleuritis is slight. It did not occur in any of these cases. Likewise postoperative dissemination of the tuberculous process did not occur. After seven and nine months, respectively, 2 cases developed signs and x-ray evidence of a tuberculous process in another lobe; both cases had lobectomies performed.

Appreciating that tuberculoma is a part of a generalized pulmonary disease, delicate surgical decision is necessary to conserve good pulmonary parenchyma and yet adequately remove the diseased tissue.

SUMMARY

A series of 48 cases of tuberculomas is presented, removed preferably by wedge resection, although segmental and total lobectomy was necessary in approximately one-third of the cases. They were removed at Fitzsimons General Hospital between Jan. 1, 1943, and March 1, 1949.

The term tuberculoma is inexact but usage has made it acceptable.

In three-fourths of the cases the lesion was discovered accidentally by routine chest roentgenograms, taken in the course of examinations for annual

branch of the posterolateral bronchus and a group of 0.2 to 0.5 cm. fibrocaseous nodules surrounded the distal end of the anterolateral bronchus. Microscopically there was calcium deposition in the caseous material clinging to the lining which is granulation tissue in some areas, fibroepithelioid tissue in others. There were fresh submilliary tubercles about and in the wall of the communication posterolateral bronchus. Other tubercles, of varying ages, were scattered in the posterolateral segment and the anterior angle.

Impression.—This was a localized area of caseous pneumonia in the posterolateral segment, which softened, established communication with the posterolateral bronchus, and ended as a cavity with tuberculous bronchitis of the draining bronchus and slight bronchiogenic spread to the anterolateral segment of the right upper lobe (Figs. 8 and 9).

REFERENCES

1. Assman, H.: Ueber eine typische Form isolierter tuberkulöser Lungen herde im Klinischen Beginn der Entwicklung, *Beitr. z. Klin. d. Tuberk.* 60: 527, 1925.
- Idem: Ueber die infraklavikulären Infiltrationen im Beginn der Lungen tuberkulose von jugendlicher Erwachsener, *Klinwchenschr.* 6: 2124, 1927.
2. Blades, R., and Dugan, D. S.: Tuberculoma of the Posterior Mediastinum, *Am. Rev. Tuberc.* 50: 41, 1944.
3. Bohrowitz, I. D.: Round Pulmonary Focus, *Am. Rev. Tuberc.* 47: 472, 1943.
4. Dorland, W. A. N.: *American Illustrated Medical Dictionary*, ed. 21, Philadelphia, 1948, W. B. Saunders Company.
5. Eßer, R., Blades, E., and Marks, E.: Problem of Solitary Lung Tumors, *Surgery* 24: 810, 1948.
6. Ghon, A.: *Primary Lung Focus of Tuberculosis in Children*, London, 1916, J. and A. Churchill, Ltd.
7. Grace, A. J.: Tuberculoma of the Mediastinum, *J. THORACIC SURG.* 12: 131, 1942.
8. Graham, E. A., and Singer, J. J.: Three Cases of Resection of Calcified Pulmonary Abscess Stimulating Tumors, *J. THORACIC SURG.* 6: 173, 1936.
- 8 a. Case No. 25432, Case Records of the Massachusetts General Hospital: Tuberculoma of lung, New England J. Med. 221: 665, 1939.
9. Haight, C., and Farris, J. M.: Tuberculoma of the Lung—Case Report, *J. THORACIC SURG.* 9: 108, 1939.
10. Holmes, G. W., and Robbins, L. L.: *Roentgen Interpretation*, ed. 7 Philadelphia, 1947, Lea and Febiger, p. 240.
11. Maurer-Mast, E., and Franklin, R. N.: Tuberculoma of the Lung, *Am. Rev. Tuberc.* 43: 301, 1941.
12. Pierson, P. H.: Solitary Foci of Tuberculosis, Development and Progress; Assman Foet Caseous Nodular Tuberculosis and Tuberculomata, *Am. Rev. Tuberc.* 45: 75, 1942.
13. Simon, G.: Sekundäre Streuherde der Lunge Insbesondere der Frühen Spitzenherde, *Engel-Pirquet Hdbch. d. Kindertuberk.* Leipzig 1: 470, 1930.
14. Webster's International Dictionary, Springfield, Mass., 1936, G. & C. Merriam Company.

DISCUSSION

DR. ELMER R. MAURER, Cincinnati.—From a total of nine pulmonary tuberculomas operated upon during the past two and one-half years, I have selected two particular case reports which I would like to present briefly because I think they illustrate well some of the hazards inherent in any program of procrastination or watchful waiting in the surgical management of these so-called quiescent tuberculous lesions (slides).

This is a posteroanterior projection roentgenogram of a 34-year-old white woman who was admitted to the hospital with a history of having been observed for several years because of this lesion which is easily apparent in the upper portion of the right lung. Approximately two to three months prior to admission to the hospital she suddenly developed a chronic hacking cough, which was unproductive and irritating in character. Repeated sputum analyses were negative for acid-fast organisms. There are numerous flecks of calcification seen. I would like to point out particularly this small strand which I think has flecks of calcification in it, connecting this mass with the mediastinum. I think this often is of help in making a differential diagnosis between tuberculous lesions and primary tumors. Lobectomy was decided upon and performed.

CASE 2.—This 23-year-old sergeant of the Air Corps stated that there was no familial tuberculous history. He had a chancre in 1943, adequately treated, with serology negative since treatment. He was in excellent health. In April, 1946, a routine chest roentgenogram during examination for separation from the service reported a 3.0 cm. dense, sharply outlined nodule in the right lung. So he was admitted to Station Hospital, Camp Beale, Calif., on April 7, 1946. Lungs were negative on physical examination. Sputa were negative for acid-fast bacilli. He was asymptomatic and afebrile. Diagnoses were pleural tumor, interlobar encapsulated fluid, and focal pneumonitis. Sedimentation rate was 27/60 on April 10, 5/60 on April 29. Transferred to Fitzsimons General Hospital June 14, 1946. Lungs were negative. Bronchoscopy was negative. X-ray examination of the chest revealed a circumscribed rounded density at level of fifth right rib axillary line. Impression was a tuberculoma or primary neoplasm. Laminagram showed it did not involve the ribs. No skin tests were made. Exploratory thoracostomy was performed and the nodule was removed.

Pathologic Report.—Specimen was a 20 Gm. wedge of lung tissue, measuring 5.5 by 4.0 by 2.6 cm. Tags of fibrous adhesions were attached to smooth glistening pleura. A 4.0 by 3.0 by 2.5 cm. fluctuant nodule occupied most of specimen. A fibrous wall, 0.05 to 0.3 cm. thick enclosed soft, semiliquid yellow caseous material; on evacuating this material the lining was trabeculated, grayish. There was a 1.0 by 0.7 by 0.7 cm. outpocket of the main nodule enclosed by a paper-thin fibrous wall. Smears for acid-fast bacilli were negative; culture was positive. Giant cells and epithelioid cells lined the caseous space; in other areas granulation tissue was present. In the outpocket there was a series of caseous foci with giant cells and submiliary tubercles in the fibrous wall, in the pleura, and pleural adhesions.

Diagnosis.—This was a completely softened tuberculoma in which the original caseous pneumonia proceeded to quick softening—an unripe dangerous type especially when enucleation is used, as it was in this case (Figs. 6 and 7).

CASE 3.—This 24-year-old Negro corporal stated that his father and two sisters had died of "lung trouble." He had no serious illnesses and no previous symptoms of chest disease. In October, 1945, a routine chest roentgenogram during examination prior to separation from service reported a rather sharply outlined density in the right upper lobe with central lucency. He was admitted to Wakeman General Hospital Oct. 29, 1945. On questioning he stated he had lost ten pounds in the past year, had night sweats once or twice a month for past three to four years. Gastric washings were positive for acid-fast bacilli. He had occasionally 99° F. afternoon temperature. On bed rest cavity decreased in size and perifocal infiltration cleared. One twenty-four hour sputum was negative for acid-fast bacilli. Sedimentation rate was 40/60. He was transferred to Fitzsimons General Hospital Dec. 29, 1945. He felt well; except for scattered râles and diminished breath sounds over right upper lobe, physical examination was negative. Fluoroscopy reported a rounded 3 by 4 cm. tuberculoma-like density in the fourth right interspace posteriorly. Temperature was normal. Sedimentation rate 10/60. X-ray examination on Feb. 5, 1946, reported cavity had enlarged. Bronchoscopy reported slight stenosis and reddening of the opening of the right upper lobe bronchus. Pneumothorax was unsuccessful due to absence of a free pleural space. A review of film in February, 1943, showed infiltration in the right first anterior interspace which had been missed. By March 10, the tiny central lucency in the nodule had enlarged to 2.0 cm. and stabilized at that size. Coccidioidin skin test 1 to 100 was 3 plus, tuberculin P.P.D. .00002 2 plus. Acid-fast bacilli found in one of three twenty-four hour specimens in January and a seventy-two hour sputum on March 13 was positive. Gastric washings were positive by culture in January and March. Sedimentation rate was 10 and 7/60. On May 2, 1946, the right upper lobe was removed.

Pathologic Report.—Specimen was the right upper lobe weighing 152 Gm.; a thin sheet of fibrous adhesions covered its surface with thick fibrous bands attached to a 2.5 by 1.2 cm. area at the upper border of a parenchymal nodule fill in the vertebral border. There was a 2.7 by 2.5 by 2.4 cm. cavity with 0.5 to 0.9 cm. thick walls in the posterolateral segment. The posterolateral bronchus was filled with caseous debris in which acid-fast bacilli were present. Soft caseous material filled the cavity. There were visible tubercles in the lower

TRANSTHORACIC THYROIDECTOMY

HERBERT D. ADAMS, M.D.

BOSTON, MASS.

INTRATHORACIC goiter may be defined as that type of goiter in which the goitrous mass lies in the mediastinum entirely below the level of the superior thoracic strait. At the Lahey Clinic nearly 28,000 patients with goiter have been operated on and hundreds of these have been classified as being truly intrathoracic by these criteria. Practically all of these goiters, many of tremendous size, have been removed satisfactorily by the cervical route. This has been technically possible chiefly by the maneuver described by Dr. Lahey of the evacuation of the semisolid colloid central portions of these masses, thereby reducing the transverse diameter of the intrathoracic mass sufficiently to permit the delivery, through the neck, of the remaining peripheral and capsular portions of the thyroid gland. This has been technically feasible and satisfactory in by far the majority of these cases.

There have been three cases in the past few years, however, in which the intrathoracic goiter has been removed transthoracically. Two were approached transpleurally because of inability to differentiate the roentgenologic findings in these cases from other types of mediastinal tumors because of their extremely low position in the mediastinum.

When the diagnosis of intrathoracic goiter has been considered, either because of the development of pressure symptoms or because a mediastinal mass has been discovered by routine roentgenologic studies, the differential diagnosis from the other common mediastinal tumors is usually difficult. For the purpose of this discussion of the differential diagnosis between intrathoracic goiter and these other common mediastinal tumors, cases have been selected in which the roentgenograms are very similar in appearance. The smaller, low-lying masses, such as in the first case (Fig. 1, *a* and *b*) may have no apparent extension upward or connection with the thyroid in the neck and may have the characteristic appearance of a dermoid cyst, bronchiogenic cyst, or even a thymoma (Figs. 2 and 3, *a* and *b*). The intermediate sized and less opaque masses (Fig. 4) are represented by the second case and have been confused at times with megaesophagus (Fig. 5, *a* and *b*), and it is wise again to stress the importance of routine barium studies of the esophagus in the differential study of mediastinal tumors. In the extremely large masses (Fig. 6) filling the entire superior mediastinum, roentgenologic studies may be identical with those of mediastinal Hodgkin's disease or arterial aneurysms of any of the great vessels originating from the arch of the aorta. The only roentgen finding differentiating intrathoracic goiter from these conditions is that in intrathoracic goiter there is usually more tracheal and bronchial displacement than in the other conditions and on fluoroscopy it can be seen to ascend and descend

Read at the Twenty-ninth Annual Meeting of The American Association for Thoracic Surgery, New Orleans, La., March 29-31, 1949.
From the Department of Surgery, The Lahey Clinic.

This is the postoperative picture; the lower lobe has filled the right hemothorax. This shows the tuberculoma which was of fair size and occupied the anterior portion of the upper lobe. Direct smear demonstrated numerous tubercle bacilli and there was a pure culture of the same organisms obtained on further study. Because of the rather sudden onset of the patient's cough—and the fact that at the time of the anatomic dissection this tuberculous mass was found to be contiguous with a major secondary branch lobar bronchus—I think we can conclude that the irritative cough was probably due to the proximity of this mass to a major bronchus and that rupture was imminent.

This 36-year-old white woman was admitted to the hospital following an attack of syncope and suggestive coronary disease at the place of her employment. Electrocardiographic tracings were entirely normal. Routine x-ray examination of the chest showed a conglomerate mass in the upper lobe of the right lung. Sputum analyses were negative for acid-fast organisms and the patient had no antecedent history whatsoever of cough, hemoptysis, or night sweats. I would like to point out the area of calcification easily seen in the lateral projection. The significance of the calcification was not appreciated until the time of operation. We had anticipated doing a right upper lobe lobectomy and, as you can see, right pneumonectomy was the final procedure. At the time of operation there was extensive calcification of the entire membranous portion of the right upper lobe bronchus, so that no point for adequate amputation and closure could be found. A pneumonectomy was decided upon and, after further examination, calcification was seen to extend up even into the trachea. The right main-stem bronchus was finally divided along an S line high on the trachea between two large calcified plaques; also there was encountered a large calcification extending into the pericardium and a portion of this latter structure was removed with the specimen.

I show this slide primarily to point out the very large calcium plaque on the bronchial stump. This next slide was following thoracoplasty and shows the mediastinum back in normal position. The patient made an uneventful recovery, and has returned to work. A pure culture of acid-fast organisms was obtained from the specimen.

I know of no accurate way in which one can determine either by roentgenographic examination or otherwise the bacterial activity of any of these masses, and I know of no way one can predict exactly when symptoms are going to occur for which reasons surgical excision should be advised.

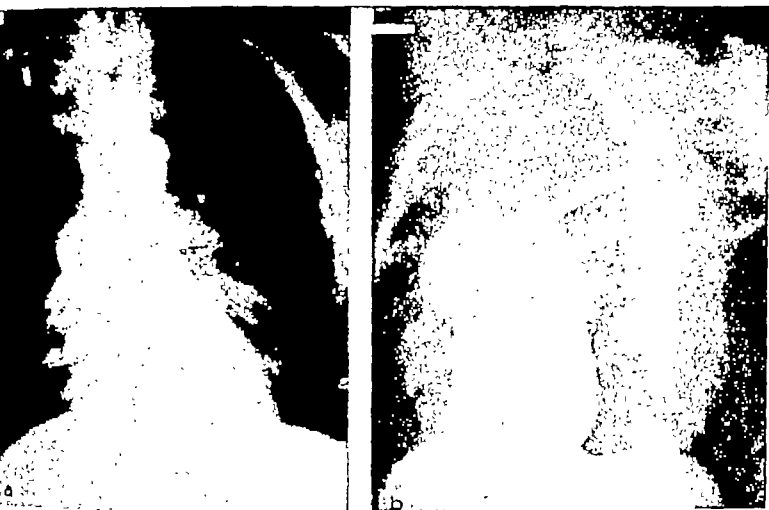


FIG. 3.—Thymoma in right superior mediastinum. a, Anteroposterior exposure; b, lateral exposure.



FIG. 4.—Intrathoracic goiter, Case 2. Larger goiter mass in right superior mediastinum, completely intrathoracic, and no displacement of trachea or esophagus.

on swallowing. Mediastinal Hodgkin's disease (Fig. 7) should always be considered and associated cervical and other nodal involvement should be carefully looked for and a biopsy done if possible. If a biopsy is not feasible and there is still a good possibility that the disease is Hodgkin's, a therapeutic



Fig. 1.—Intrathoracic goiter, Case 1. *a*, Low-lying discrete adenomatous goiter in right superior mediastinum. *b*, Bucky film of same, showing slight tracheal deviation to left.



Fig. 2.—Bronchiogenic cyst in right superior mediastinum similar in appearance to goiter shown in Fig. 1.

x-ray treatment may be given, with a rapid regression of the size of the mass being accepted as diagnostic. However, we are in favor of the growing trend toward advising exploration in order to obtain accurate pathologic diagnosis in all of these conditions.

If there are clinical findings such as a bruit over the upper mediastinum, abnormal pulsations in the neck, or altered blood pressure in the upper extremity, or fluoroscopic evidence that this mass could be a vascular lesion (Fig. 8), it is best to carry out angiography to make certain of the diagnosis and determine accurately what vessel is involved. Without this preoperative knowledge and the proper approach, an aneurysm of this type can be easily ruptured during exploration. Such a study would entail retrograde arterial catheterization and an angiogram. Neurofibromas and ganglioneuromas are



Fig. 8.—Aneurysm of the innominate artery.

common at this level in the mediastinum but lie paravertebrally in the lateral exposure (Fig. 9, *a* and *b*). Teratomas often lie in the same mediastinal plane as goiter, but frequently can be differentiated by demonstrating roentgenologically the presence of bone or teeth in one of the exposures (Fig. 10, *a* and *b*). Having considered all of these possibilities and having done what studies are indicated and possible, however, the differential diagnosis may still be undetermined at the time of exploration.

The larger masses produce lower tracheal and main-stem bronchial compression and the symptoms produced may progress to serious proportions. The third patient was experiencing serious obstructive symptoms and it is well to



Fig. 5.—Mega-esophagus. Mass in right upper thorax and mediastinum simulating closely that shown in Fig. 4. *a*, Anteroposterior exposure; *b*, lateral exposure.

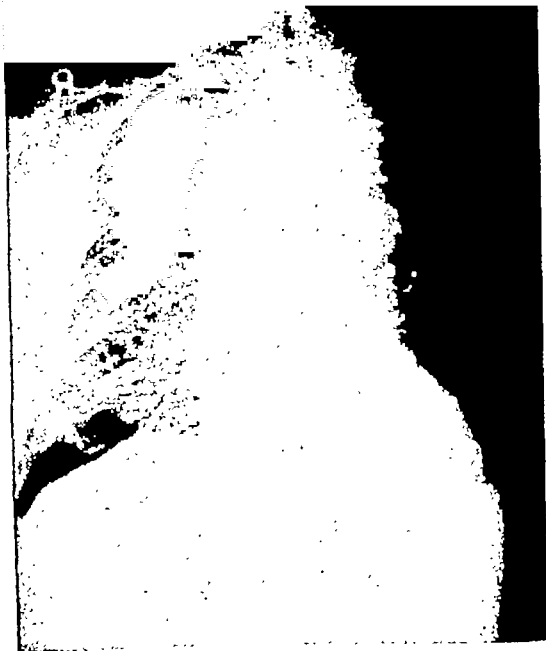


Fig. 6.

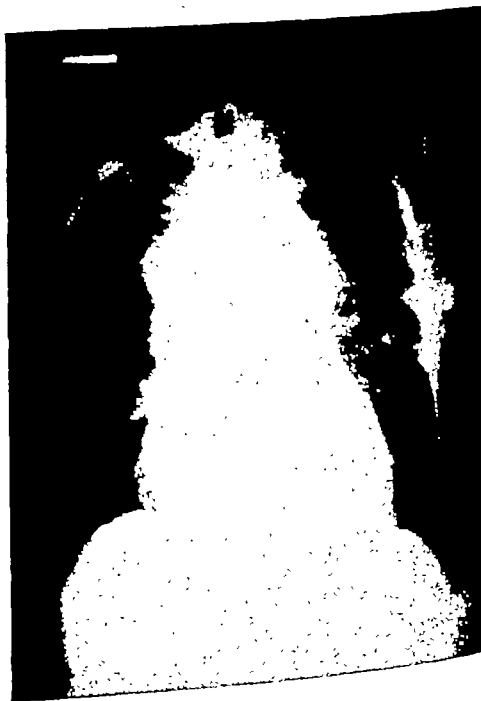


Fig. 7.

Fig. 6.—Large intrathoracic goiter with marked tracheal deviation to the right, removed by the cervical route.

Fig. 7.—Mediastinal Hodgkin's disease.

remember that such an obstruction associated with a true, completely intrathoracic goiter cannot be relieved by an emergency tracheotomy since the obstruction is below that point. If the patient is allowed to progress to a critical point of lower tracheal collapse and complete obstruction, it will be too late to do any type of surgery that will be sufficiently rapid to save the patient's life. In these tremendous goiters of this type (Fig. 11, *a* and *b*), the trans-thoracic resection is a long and hazardous procedure. This is due primarily to their extensive vascularity and their close relationship to the major mediastinal structures. This third patient was operated on by the transpleural approach because of marked obstructive symptoms and because an attempt had previously been made elsewhere to remove the goiter through the neck, with failure, owing to severe hemorrhage encountered and to its tremendous size.

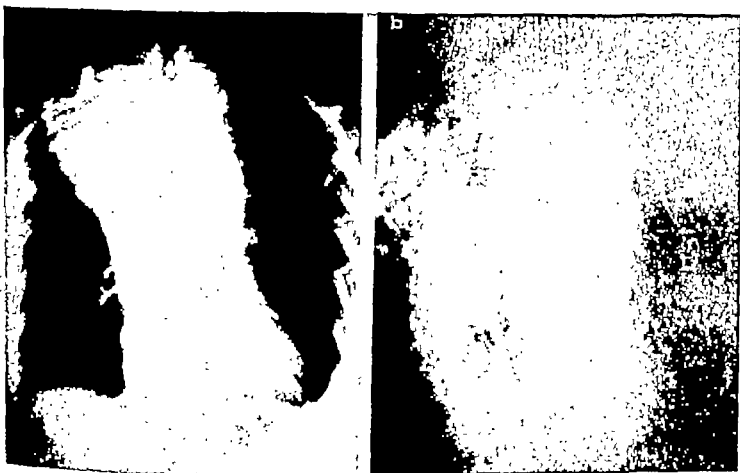


Fig. 11.—Intrathoracic goiter, Case 3. *a*, Tremendous goiter mass filling superior mediastinum and right upper thorax. *b*, Lateral exposure showing the anterior position of the mass.

The anesthetic must be administered by skilled and experienced anesthesiologists who will be able to cope with almost any type of respiratory obstruction or circulatory collapse throughout this lengthy procedure. Intratracheal anesthesia is used, as in all other types of thoracic surgery.

A posterolateral approach is made, resecting the sixth rib and sectioning the fifth and seventh ribs near their transverse processes, giving a wide exposure of the upper thorax. This transpleural approach is done through the right thorax since the greater proportion of the mass extends into this thorax and its mediastinal extension can be dissected under better vision without encountering the aortal arch which would obscure the exposure from the left



Fig. 9.—Medialastinal neurofibroma. *a*, Anteroposterior exposure; *b*, lateral exposure showing posterior paravertebral position of mass characteristic of this disease.



Fig. 10.—Medialastinal teratoma. *a*, Anteroposterior exposure; *b*, lateral exposure showing diagnostic bone and teeth within the mass.

It became obvious that some other method of increasing the mediastinal exposure would be necessary if this large mass was to be removed without a fatal hemorrhage. To accomplish this, the azygos major vein was doubly ligated and sectioned, permitting the superior vena cava to be retracted farther anteriorly, and the cleavage plane of the anterior surface of the goiter was then established. There was extensive vascularization at both the superior pole, which is the usual source of the blood supply of intrathoracic goiter, and the inferior pole, with marked venous sinusoidal vascularization of all surfaces. So far as possible the blood supply was controlled under direct vision, but in its more extreme extensions to the left side of the mediastinum and behind the arch of the aorta the cleavage planes had to be followed blindly by blunt dissection with the fingers. Severe venous hemorrhage was encountered and even with the mass finally out of the way, it was impossible to secure this bleeding by clamping, tying, or suturing. It was finally secured with five Oxycellulose packs, measuring 3 by 3 inches. The mediastinal pleura and the thorax were closed, leaving an intercostal catheter for suction drainage of the pleura.

The patient made a good recovery, the lung re-expanded promptly, and there was no late effusion. On the tenth day, however, the patient began to run a septic type of fever, with slight leucocytosis, and by roentgenologic examination the mediastinal shadow remained widened. After some days mediastinal pressure symptoms developed and she was therefore taken to the operating room and a large mediastinal abscess drained through the supra-sternal notch. The culture was hemolytic *Staphylococcus aureus*, an infection which was almost certainly superimposed upon a hematoma and the large quantities of foreign material, the Oxycellulose, that was used within this mediastinal space. For these same reasons it is very probable that it would have been good judgment to have drained the mediastinum through the neck at the close of the operation.

CASE REPORTS

CASE 1 (Fig. 1).—The patient was a man, aged 53 years, who had had no symptoms. A mediastinal mass was discovered on routine x-ray chest survey of employees. Physical examination was essentially negative. The roentgen report was of a circumscribed mass to the right of the upper mediastinum and slight deviation of the trachea toward the left. It was elevated on swallowing and did not interfere with the esophagus. There was also enlargement of the left hilar region. The findings were consistent with lymph node enlargement, probably Hodgkin's disease.

At operation, the sixth rib was excised subperiosteally and the fifth and seventh sectioned posteriorly. An adenomatous goiter mass, 7 by 5 by 3.5 cm., lying in the angle between the trachea, superior vena cava, and azygos major vein, was excised. Some large paratracheal lymph nodes were also excised. The pathologic report was multiple colloid adenomatous goiter (70 Gm.) and tuberculous adenitis. The patient made an uneventful recovery.

CASE 2 (Fig. 4).—The patient was a woman, aged 46 years, who had had no symptoms. During a routine check-up examination by the family physician, a roentgenogram of the chest showed a shadow in the right lung field, and further investigation was advised. The

side throughout the greater part of the dissection. It is also most probable that it is the aortal arch that limits the extension to the left thorax to some extent. These goiters have extended behind the arch but not often lateral to it. In the huge goiter removed in this manner (Fig. 12), the portion of the mass extending into the upper right thorax was freed subpleurally without much difficulty, with the exception of the management of extensive venous sinuses over the entire surface. The mediastinal portion of the mass was wedged

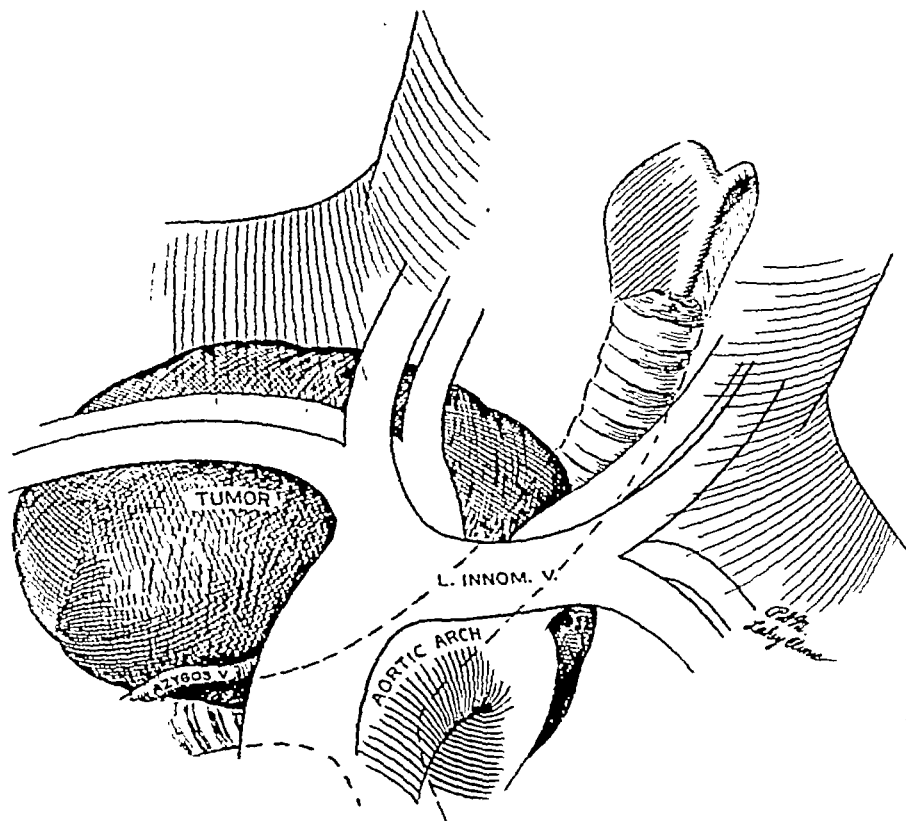


Fig. 12.—Anatomic sketch showing relationship of the intrathoracic goiter (Case 3) to the great vessels and trachea.

firmly in a plane with the lower trachea, carina and main-stem bronchi posteriorly, and the great vessels anteriorly. The right mediastinal exposure showed the mass bulging from behind the superior vena cava and the azygos major vein, compressing these structures anteriorly. The goiter was of such bulk and wedged in so firmly between the planes of the lower trachea and the great vessels that it was impossible to deliver it from the mediastinum safely. In an attempt to apply Dr. Lahey's principle that the center of the mass could be evacuated, thereby reducing the bulk of the mass sufficiently to deal with its capsular remnants more effectively, the mass was broken into with the finger. However, its central portion was of too solid consistency to make this maneuver possible or to reduce the bulk of the mass by evacuating its central portion. In addition to this, marked hemorrhage was encountered within the mass, necessitating packing with Oxycellulose gauze in order to control the bleeding.

tion of it, but in addition she had this huge mass here, in the right mediastinum and a small one in the left chest wall. Symptoms were those of mediastinal tumor. The first thing we did was to remove this small, chest wall, tumor mass which proved to be an extrapleural, encapsulated, neurogenic tumor. Several months later the mediastinal tumor was operated on through a posterolateral incision and a huge mediastinal adenomatous goiter was removed. There was no communication between this and the substernal goiter which lay in front of it. You can see from the specimen that it is completely encapsulated above and the substernal goiter came down in front of it.

DR. ARTHUR S. W. TOUROFF, New York.—I would like to point out that since radio isotopes have become available, the diagnosis of intrathoracic goiter can be made in practically 100 per cent of cases. During the past year, our Department of Medical Physics has studied thirty-four anterior and superior mediastinal tumors, of which twenty-one were proved to be thyroid tumors. Twenty of the twenty-one gave positive results on radioactive iodine "tracer studies." The radiologic and clinical diagnoses in these cases included such lesions as aneurysm of the aorta, neurofibroma, dermoid, and esophageal dilatation. There was one case in which an incorrect diagnosis was made. This was a case of very thin-walled colloid cyst in which there was no functional thyroid tissue present. In any mass in which functional thyroid tissue is present all that is necessary to make the diagnosis is to give the patient a dose of radioactive iodine and then pass a Geiger counter over the region of the mass. As soon as one hears the characteristic "clicks," the diagnosis is made.

DR. DERYL HART, Durham, N. C.—I want to report a recent operation, the patient still in the hospital, primarily to urge that if the surgeon is certain that he is dealing with an intrathoracic thyroid which cannot be removed through the neck or if he cannot rule out a thyroid deriving its blood supply from the neck, that the patient be in such a position that the surgeon has ready access to the neck and the chest. It is hard for me to see how the blood supply originating from the thyroid vessels can be controlled through the thoracic approach.

Our patient, aged 70 years, had an intrathoracic tumor, which in seven years had increased only 2 cm. in diameter. On admission she had an intractable cough, complete paralysis of the right vocal cord, almost complete paralysis of the left, and was suffering from severe respiratory embarrassment. It was our impression that the lesion was benign, probably thyroid, possibly a dermoid, but definitely not an aneurysm. Because of the size of the tumor and in the absence of a palpable mass in the neck, it was approached through the right chest, but with the neck exposed. We found it to be thyroid lying posterior to the great vessels. Because of its location and size, I do not see how it could have been removed satisfactorily by splitting the sternum, but it was readily accessible by an anterior right transpleural approach. The tumor in the chest was dissected free and it was determined that there was a pedicle extending into the neck and that all of its blood supply came from the neck.

Through the usual thyroid incision the enormous superior pole vessels were divided and ligated. The thyroid isthmus was split but it was impossible to expose the inferior thyroid vessels or the lateral vein, since the thyroid was anchored to the mass in the mediastinum which prevented delivery or rotation of the thyroid pedicle in the neck. By applying pressure to the mass within the chest and traction on the pedicle in the neck the mass was delivered. This procedure avoided tension on the blood supply. After the mass was delivered, further operative procedure was as for any large thyroid. By this double approach we had the blood supply under control at all times. Her respiratory obstruction was relieved immediately. We have not as yet examined the vocal cords for return of function, but she is making an entirely satisfactory convalescence.

DR. FREDERICK G. KERGIN, Toronto.—There seem to be three groups of patients coming within this general group; one is the patient who has the obvious goiter which is intrathoracic and which it is possible to remove from the neck; but if one fails to do so one may then split the sternum and remove it by combined approach (slide).

radiologist's report was a lobulated soft tissue mass in the right upper thorax extending out from the mediastinum anteriorly. It did not press on the esophagus or trachea. It could have been an unusual mediastinal thyroid adenoma but was more suggestive of a dermoid.

At operation, a right posterolateral transthoracic approach was made. An adenomatous goiter mass, 9 by 8 by 5 cm., lying above the hilus of the lung, was excised from the mediastinum. The entire blood supply entered at the inferior pole of the mass. The pathologic report was multiple colloid adenomatous goiter; it weighed 190 Gm. The patient made an uneventful recovery.

CASE 3 (Fig. 11).—A woman, aged 53 years, had the chief complaint of rapidly increasing difficulty in swallowing and breathing. Thyroidectomy had been attempted twenty years before entry. Two years previously a substernal goiter was found on roentgen examination. She was told following the previous operation that it was impossible to remove all of the goiter because of severe hemorrhage and the tremendous size of the intrathoracic extension. On physical examination there was moderate stridor and engorgement of the superficial veins of the upper thoracic and cervical region. The radiologist's report was as follows. In the upper mediastinum anteriorly there was a large, sharply circumscribed mass which filled the entire upper third of the right thorax and extended across the midline to the left behind the aortic arch. There was no significant deviation of the esophagus or trachea. This mass showed no pulsation. Impression was a large mass in the anterior mediastinum and right upper thorax with some extension to the left. Lymphoma had to be considered.

The operation and clinical course were as described previously. The pathologic report was multiple colloid adenomatous goiter, measuring 18 by 8.5 by 7 cm. The specimen weighed 700 Gm.

In conclusion, completely intrathoracic goiter is common and even very large masses up to 1,000 Gm., in a large majority of cases, can be removed through the neck by employing Dr. Lahey's maneuver. However, in not all of these cases is it technically possible to use this approach and these will require transthoracic resection. Others that lie particularly low in the mediastinum will also require excision transthoracically because they cannot be distinguished from other types of mediastinal tumors.

The technical problems involved in transthoracic excision of these extremely large completely intrathoracic goiters are chiefly those of obtaining adequate mediastinal exposure and control of the extensive blood supply. Drainage of the mediastinum through the suprasternal notch is advisable.

DISCUSSION

DR. RICHARD H. MEADE, JR., Grand Rapids.—I want to congratulate Dr. Adams for his excellent presentation of this problem, and to emphasize the fact that in certain cases it is impossible to control the blood supply to a substernal goiter through the neck (slides).

This first patient had an ordinary goiter which troubled her a good deal. X-ray examination of the chest showed this extension below. Because she was more concerned with the cervical goiter than the mediastinal one, a combined operation was done with removal of the cervical goiter through the usual incision and the other through splitting of the sternum. It was thought at first that the goiter was continuous, both in the neck and the mediastinum, but we found when we dissected the specimen that they were two separate tumor masses getting their blood supply from two different areas. They were joined by a fibrous membrane. There was no apparent communication between the vessels.

In the next case the problem was very different; here the problem was one of diagnosis. She had a goiter of 34 years' duration, and we knew there was some sternal prolunga-

the operation was dead. Another doctor in the area wrote to me and said he remembered the case and that he felt she was definitely toxic at the time. One wonders whether there had been any compensatory enlargement of this particular nodule following removal of the goiter in the neck.

The post-mortem case, which Dr. John Fisher of our pathologic department recalled for me, was a woman 66 years of age who had died of cerebral hemorrhage. She had a large distinct mediastinal mass situated low down. She also had a history of having a subtotal thyroidectomy years before for a toxic thyroid, but there was no detailed history with regard to that.

I wonder whether any of the other cases reported—Dr. Adams' cases for instance—have had previous thyroid operations, and what possible significance that might have. It seems strange that both the examples we have encountered have had previous operations performed.

DR. ERNEST JAMES, Hamilton, Ont.—This is a report of a patient presented before this Association in 1938. At the age of 28 years, in 1930, she had a thyroidectomy performed because of a toxic adenoma. At that time one adenoma about the size of an orange, retro-sternal, was removed through the cervical route. She presented herself again in March, 1938, complaining of dyspnea and tachycardia which had been coming on for about five months. The thyroid was not enlarged. X-ray examination of the chest showed the right side to be opaque, but after removal of 2,000 c.c. of rather blood-stained fluid and replacing it with air, it was possible to demonstrate a large tumor mass in the anterior mediastinum, which was protruding somewhat toward the right. A definite diagnosis was not made but because she appeared slightly toxic she was Lugolized, and in November, 1938, operation was carried out. The approach was made by removing about one inch of the second rib anteriorly, separating the parietal pleura, and then dividing the third and fourth costal cartilages. The ribs were then cracked in the anterior axillary line and a window in the chest thus opened. The parietal pleura was stripped out without tearing it, and an oblong tumor was shelled out from the anterior mediastinum which measured about 6 by 4 inches; a second small one, about 1.5 by .5 inches was removed. No blood supply was encountered, there was very little bleeding, and no blood vessels had to be dealt with (slides).

This slide is shown to demonstrate the size of the tumor, with the smaller tumor lying next to it. The microscopic section shows it to be thyroid tissue.

Whether this is a tumor which migrated from the neck into the chest or whether it should be called aberrant thyroid I do not know. I think the absence of any demonstrable blood supply suggests that it might possibly be an aberrant thyroid.

DR. ADAMS.—I appreciate very much these interesting comments. Briefly, with regard to diagnosis I think Dr. Touroff's suggestion of using radioactivated iodine with a Geiger counter would certainly be a very definite and valuable addition to our diagnostic methods.

With regard to whether these completely intrathoracic goiters are truly aberrant, or whether they have detached themselves from the thyroid in the neck and descended into the mediastinum, I believe it will be difficult to decide definitely as to their origin in any given case. I think, in addition to that, one must be prepared to encounter and to deal with almost any type of vascularity; some of these have had their full supply from the inferior pole, and, as we know, in the majority the major blood supply comes from above. I think it would depend largely on the size and consistency of the intrathoracic extension whether or not you felt you must extend the incision into the thorax. In the medium-sized ones I am sure that splitting the sternum will give very satisfactory exposure. In the extremely large ones I believe the best approach is the right posterolateral transthoracic approach.

One of these three patients had had a previous thyroidectomy; that was the one whose roentgenogram simulated mega-esophagus in appearance, and it was completely unconnected

This is such a case; due to adherence of the thyroid in the chest and mediastinum, we could not remove it safely, so the sternum was split and it was then removed with ease. The next group are those in which one knows the nature of the tumor, and elects to do a transthoracic approach. Here is a patient who obviously had a goiter lying behind the trachea, a patient of Dr. Robert Janes; in which he elected to do a right transpleural approach and found that the operation was accomplished with ease. Again, there are patients in whom the thyroid presents as an intrathoracic or mediastinal tumor, in which one can suspect its nature but cannot reach it through the neck. In the next case, the tumor lies in front of the heart, with calcification. We diagnosed this as an intrathoracic ectopic goiter, and did a transpleural approach from the right side. The next slide shows a similar case in which the goiter lies in the anterior mediastinum, again with calcification, on which we have made this diagnosis but have not yet removed the goiter. I think that with this group there is no chance of reaching it through the neck, and primarily a transthoracic approach must be made.

DR. E. E. AVERY, Chicago.—I had two questions to ask Dr. Adams; the first on how to predetermine the diagnosis of substernal thyroid was answered very nicely by Dr. Touroff. In the future, we shall remember to use radioactive iodine in the differential diagnosis of superior mediastinal masses. The second question deals with the aberrant blood supply to these tumors, and I have two cases from Hines Hospital to illustrate this problem. The first slide reveals a right superior mediastinal mass. The work-up gave no particular indication that this was thyroid tissue. The approach was by the transthoracic route and a large adenomatous thyroid was found which derived its major blood supply from the right subclavian artery.

In the second case, the chest film was similar. The thyroid gland was not enlarged to palpation and the basal metabolism rates were normal. Exploration was done through the transthoracic route, and in this case the frozen section revealed adenocarcinoma of the thyroid. The blood supply to this tumor was derived both from the inferior thyroid artery and a large branch from the right internal mammary.

I want to ask Dr. Adams how often aberrant thyroids were found in the mediastinum in this large series of cases, and what vascular supply has been found to these aberrant thyroids? Perhaps a clue to this answer might be in the morbidity and mortality due to severe hemorrhage following removal of these thyroids through the neck. It seems to me that severe bleeding could be encountered if one of these vessels were torn.

The transthoracic approach is not ideal because the major blood supply is normally derived from the inferior thyroid vessels and the tumor usually extends up into the neck, however, the procedure can be continued through the usual thyroid incision after the transthoracic portion has been completed. Perhaps a more direct attack could be made by the usual thyroid approach and then split the upper portion of the sternum to expose the superior mediastinum.

DR. A. J. GRACE, London, Ont.—There is one point I would like to bring out in presenting one operative case and one post-mortem case in about 3,000 post-mortems at our hospital. The operative case was typical of several that have been presented (slide). The patient was a woman 33 years of age, whose work-up had been done by Dr. F. S. Kennedy—I was called in to operate for an undiagnosed mediastinal tumor. The shadow seen is typical of several that have been shown; after removal it measured about 2 by 2 by 2.5 inches and weighed 50 Gm., and was diagnosed pathologically as a colloid adenoma. There was no connection between it and the tissue above that we could discover. It seemed to be surrounded by areolar tissue, and to lie against the lowest part of the trachea on the right side, immediately against the upper part of the azygos vein.

There was one point in this woman's history; she had had a subtotal thyroidectomy done in a smaller hospital four and one-half years previously. There was no history obtainable; that hospital had burned down when I wrote for records, and the doctor who had performed

peritoneal canals, communicate caudally with the primordium of the peritoneal cavity. Ventrally the borders of the cavities are limited by the primitive septum transversum into which the liver grows and through which the primitive veins enter the sinus venosus of the heart.



Fig. 12.—Section of a 30 mm. human fetus showing beginning fold in the medial wall of the pleural cavity.

As the embryo grows, the lungs begin to form as definite projections from the ventral wall of the primitive pharynx. Coincident with the beginning of the lung buds, the pulmonary ridge, a fold of tissue lying internally to the common cardinal veins at their junctions with the lateral ends of the septum transversum, becomes divided into two folds. The fold growing mesially, ventral to the developing lung, cuts off the pleural cavities from the pericardial cavity by forming the median part of the pleuropericardial membrane. This occurs during the 5 to 10 mm. stage of development. That part of the fold caudal to the lungs grows mesially and closes the communication between the pleural and peritoneal cavities. As a result of these changes, the lungs lie in the primordia of the dorsal parts of the future pleural cavities. These folds, which separate the pericardium from the pleural cavities and the pleural cavities from the peritoneal cavity, are free growing and do not invade semi-solid connective tissue to form cystic spaces.

However, beginning at about the 15 mm. stage, the lungs enlarge and with this enlargement the ventral ends of the primitive pleural cavities begin to grow down into the mesenchyme of the lateral body wall between the mesothelium of the pericardial cavity and the ectoderm of the primitive thorax.

Treatment.—Because of the many difficulties in preoperative differentiation of intrathoracic masses, exploration should be carried out unless clinical evidence points to a hopelessly inoperable condition. This viewpoint is widely shared by many writers. Harrington,¹⁰ as early as 1929, had removed nine benign tumors and had no deaths or ill effects from the operations. He advocated at that time complete removal of benign tumors in the thorax before they could undergo malignant degeneration or cause death from pressure.

Alexander¹ cautioned against delay in removing intrathoracic tumors and lay particular stress on the circumscribed neoplasm which many physicians assume to be benign and hesitate to remove.

Heuer and Andrus,¹¹ in a survey of their cases, found that the benign cysts lend themselves to surgical removal and that the size of the tumor is unimportant since very large tumors, though they present mechanical problems, are quite often easily removed with little effect on the patient.

There are three reasons for exploring all intrathoracic masses unless obviously malignant and at an inoperable stage. In the first place, no mass can be confidently considered benign without a biopsy. In the second place, the mechanical effects of a space-consuming lesion on the physiology of respiration and cardiac action must eventually become important and be expressed in symptoms. In the third place, some benign lesions, such as dermoid cysts, may cause complications later by rupture into the bronchus or the pleura. It must be remembered that an accurate pathologic diagnosis cannot be made without exploration. The safety of modern intrathoracic surgery makes this reasoning all the more valid.

Etiology.—At least two theories of pathogenesis have been advanced. Lambert¹⁵ pointed out that during the development of the pericardium there appears early a series of disconnected lacunae in the mesenchyme both lateral and central to the primitive streak. As the embryo develops, these lacunae enlarge, gradually merge, and condense to form the pericardial celom which persists temporarily as an independent cavity. Eventually a connection is formed between it and the pleuroperitoneal celom from which the pleural and peritoneal cavities develop. The ligamentum transversum and ducts of Cuvier close the connection between the pericardial celom and pleuroperitoneal celom, and also divided the pleuroperitoneal celom into pleural cavities above and the peritoneal cavity below. Failure of these normal processes of development have been noted such as the familiar congenital hernia of the diaphragm which results from failure of the ligamentum transversum to divide the pleuroperitoneal celom into pleural and peritoneal cavities. Lambert contended that these reported cysts are probably due to the failure of one of the primitive lacunae to merge with the others and its persistence and development into an independent cavity, forming a cyst. Lambert therefore designated these lesions as "pericardial celomic cysts" and Laipply¹³ agreed.

Another explanation of these cysts is suggested by Kindred.¹² He pointed out that the dorsal parts of the pleural cavities are derived from the original dorsal regions of the primitive pericardial cavity. These regions, the pleuro-

Nomenclature.—It is always confusing in the discussion of pathologic lesions when uniform terminology is not employed. In the present instance, this fact is amply demonstrated. In naming a lesion of presumably congenital origin the probable source of the abnormal tissue must be considered. The reason for multiple names for these mediastinal cysts lies in the failure of agreement on pathogenesis. Kindred's explanation of their origin seems a reasonable one. We, therefore, suggest that a name be chosen which recognizes this contribution. The name "mesothelial mediastinal cyst" fulfils this requirement. It is urged that this name be adopted in future reports of this disease.

REFERENCES

1. Alexander, John: J. A. M. A. 119: 305, 1942.
2. Cabot Case No. 23492: New England J. Med. 217: 24, 1937.
3. Cabot Case No. 26291: New England J. Med. 223: 105, 1940.
4. Corri, A. R., and Gale, J. W.: Ann. Surg. 113: 1941.
5. D'Abreu, A. L.: Brit. J. Surg. 25: 317, 1937.
6. Dufour, H., and Mourrut: Bull. et mém. Soc. de med. de hôp. de Paris 53: 1482, 1929.
7. Edwards, Tudor: Brit. J. Surg. 14: 607, 1927.
8. Freedman, E., and Simon, M. N.: Am. J. Roentgenol. 35: 53-56, 1936.
9. Greenfield, I., Steinberg, I., and Touroff, A.: J. THORACIC SURG. 12: 495, 1943.
10. Harrington, Stuart: Arch. Surg. 19: 1679, 1929.
11. Heuer, G. J., and Andrus, W. D.: Am. J. Surg. 50: 146, 1940.
12. Kindred, J. E.: Personal communication.
13. Laipply, T. C.: Arch. Path. 39: 3, 1945.
14. Lam, Conrad R.: Radiology 48: 239, 1947.
15. Lambert, Adrian: J. THORACIC SURG. 10: 1-7, 1940.
16. Leahy, L. J., and Culver, G. J.: J. THORACIC SURG. 16: 695, 1947.
17. Plekhardt, O. C.: Ann. Surg. 99: 814-816, 1934.
18. Weber, Parkes: Brit. M. J. 1: 851, 1934.

DISCUSSION

DR. GEORGE F. SKINNER, Saint John, New Brunswick.—I have been intensely interested in this discussion, because in October, 1930, in the Journal of this Association, we reported a case obviously similar to this one, under the title of "Mediastinal Cystic Lymphangioma." We were not entirely satisfied with this diagnosis in spite of the fact that it was supported by several pathologists including Tumor Registry. At that time we could find no similar case in the American literature although the possibility of such a tumor was mentioned. There were a few reports in the German literature.

A 4-year-old boy was admitted on account of respiratory embarrassment. The entire right side of the chest was filled. After aspirating the pleural fluid, two-thirds of this side was seen to be filled with a multiloculated cystic tumor. On exploration the tumor was found to be composed of numerous cysts ranging in size from 1 mm. to 10 cm. It was easily separated from the upper mediastinum, the vena cava, the pericardium, and from the lateral portion of the diaphragm, but in the angle between the pericardium and the diaphragm very small cysts appeared to infiltrate into the tissues. In spite of being forced to leave small cysts in this angle the boy was well and showed no sign of recurrence when examined ten years after the operation.

DR. CONRAD R. LAM, Detroit.—I should like to present a case which will show the development of such a cyst, in a patient 28 years old at the time of this roentgenogram, who was operated on ten years after these films were made (slides). Three years later, the shadow had increased in size and a diagnosis of congenital heart disease was made. Later, the patient was kept in bed because she was presumed to have a tremendously enlarged heart. The cyst was easily removed through a right sixth interspace incision.

Primordia of the ribs and intercostal muscles grow into the mesoderm lateral to the enlarging pleural cavities. Internally, the mesothelium of the pleural cavity comes into close contact with the mesoderm which is external to the mesothelium of the pericardial cavity. Thus, the enlarging ventral parts of the pleural cavities meet opposition as they grow. From such a method of growth, it is conceivable that unequal areas of enlargement may occur and folds may be present in the wall of the advancing pleural cavities. Such folds may be obliterated with later growth, others may enlarge at the same rate as the growing pleural cavity. When the latter occurs, it is possible that such folds may be cut off and form cystic sacs around which connective tissue can be laid down in the same manner of the lamina propria of the pleura. A morphologic condition which lends evidence to this view has been observed in the medial wall of the pleural cavity of a 30 mm. human fetus (Fig. 12). This fold is lined with simple squamous epithelium and is almost cut off from the parent cavity. It forms a narrow fold which causes the surrounding mesenchyme to project into the parent cavity along its ventral lip. If this condition persists, a saclike cyst would develop and could project either into the pleural cavity or the pericardial cavity. Such cysts could occur in any region of the pleura which has been derived from the secondary invasion of the body wall by the pleura. The distribution of the cysts reported have always been found in regions which have passed through morphologic changes during development as described previously.

There seems to be no necessity for assuming that the cysts are derived from persistent infracardiac bursae or their abnormal outgrowths, since cysts derived from such sources would lie in the region of the esophagus, at the central tendon region of the diaphragm. Nor does it have to be inferred that these cysts are remnants of unabsorbed vesicles from which the primitive pericardial cavity is formed. The latter are too delicate and occur too early in development for contribution in the formation of such cysts.

The morphologic evidence available, therefore, suggests that these cysts originate by aberrant growths from the mesothelium of the pleural cavity as it invades the mesenchyme of the body wall. Whether or not hydrostatic pressure contributes to their formation is not known since no measurements have been made of the fluid in the primitive pleural cavities. It has been suggested in the previous discussion that with the separation of the pleural cavities from the pericardial cavity, the pleuropericardial membrane is growing into a free space and is not likely to have its free growing margin modified. However, if the pleuropericardial membrane should grow more rapidly than normal and fold on itself before it fuses with its fellow to form the completed pleuropericardial wall, it is conceivable that a sac could form posterior to the heart in the regions of the great vessels and the bronchi. This region is so compressed during development and the subjacent mesenchyme undergoing so much differentiation into the cartilage of the bronchi and the surrounding musculature, the cysts in this region should be relatively rare. Lambert's theory seems less easy to accept.

INTERCOSTAL INCISION IN TRANSPLEURAL OPERATIONS

JOSEPH A. WEINBERG, M.D., AND (BY INVITATION) ALVIN R. KRAUS, M.D.
VAN NUYS, CALIF.

WIDENING of the scope of thoracic surgery has directed attention to the lessening of morbidity as well as mortality. The type of incision for approach to the thoracic cavity, though seldom viewed as a major problem, has an important bearing on the morbidity attending thoracic operations.

In almost all surgical procedures in which the pleural cavity is entered, it is the practice to remove one or more segments of rib or at least to divide one or more ribs posteriorly. This method has been responsible for much of the intercostal neuritis, pneumonitis, and pleural effusion that is so common in any large series of thoracic operations.

In an effort to lessen morbidity, shorten the time of operation, and preserve the normal contour of the thoracic wall, we have used the intercostal incision in a variety of indications for thoracotomy. The results have been impressive. This type of approach has been found to be universally applicable and is now used routinely by us in pneumonectomy, lobectomy, segmental pulmonary resection, drainage of acute empyema and pericardial empyema, decortication of the lung, repair of the diaphragmatic hernia, transthoracic vagotomy, and transdiaphragmatic splenectomy. It has also been used in resection of the proximal two-thirds of the stomach and distal esophagus. Other members of our staff have used this same approach in thoraco-lumbar sympathectomies.

The advantages of the intercostal approach are more than facilitation of opening and closing the thoracic wall. A comparison with cases in which resection or posterior division of a rib was performed reveals a definite difference in morbidity, with a lower incidence of pneumonitis, pleural effusion, and post-operative intercostal neuritis with intercostal incision. A further advantage to the patient is the preservation of the normal contour of the thorax.

Intercostal pain is almost a constant factor with costal resection for exposure of the pleural cavity. This is due to irritation of the intercostal nerve by the sharp edge of the cut rib. Grimson,¹ in a report on 144 thoracotomies in which ribs were resected, stated that all patients had pain in the chest, which was most intense during the second week after operation. This intercostal neuritis is such a common sequel to thoracotomy that some surgeons crush the intercostal nerves above and below the incision² or inject them in order to lessen postoperative pain. In Grimson's series, the intercostal nerve inferior to the incision was routinely excised or avulsed and in some patients the intercostal nerve adjacent to the incision was crushed; in others they were injected with alcohol. None of these procedures effected definite relief. In addition to being an objectionable feature per se, the pain also tends to cause splinting of the

From the Department of Surgery, Birmingham Veterans Administration Hospital.

Published with the permission of the Chief Medical Director, Department of Medicine and Surgery, Veterans Administration, who assumes no responsibility for the opinion expressed or conclusions drawn by the author.

Read (by title) at the Twenty-eighth Annual Meeting of The American Association for Thoracic Surgery, Quebec, Canada, May 31, June 1, 2, 1948.

With regard to the suggestion that there be a change in the nomenclature, I call attention to the fact that these cysts are close to if not attached to the pericardium and someone may suggest that they be called "para-pericardial" cysts.

DR. F. A. HUGHES, JR., Memphis, Tenn.—I would like to mention that the thoracic surgery staff at Kennedy General Hospital (Army) removed 14 of these cysts. One was in the left upper mediastinum. Twelve were diagnosed on routine x-ray examination and were asymptomatic. This group formed 20 per cent of the benign mediastinal tumors removed by the Army group. None of the cysts showed a connection with the pericardial space.

DR. GUSTAF E. LINDSKOG, New Haven, Conn.—We have seen two of these anterior cystic lesions in the last year; one multilocular and one unilocular. They show in the wall definite histologic evidence of smooth muscle tissue and a mesothelial type of lining. I do not believe the pericardium contains any smooth muscle; if they were of pericardial derivation, one would not expect that they would show smooth muscle. These were deeply attached in the anterior costophrenic angle, and we suspect their origin from anterior, lymphatic channels.

DR. PLIMPTON GUPTILL, Rochester, N. Y.—I would like to show a couple of slides showing findings very similar to those Dr. Drash showed. These were two men in the middle fifties, asymptomatic (slides).

There is anterior displacement of the esophagus. There is a tumor 8 cm. in diameter with a small projection on the right side.

It has been suggested by Mallory that they may have had an epithelial lining which was destroyed by pressure of the contents. The fluid content in both these cases was clear; chemical analysis was not made.

DR. FRANK B. BERRY, New York.—I have had two of these cases and the structure of both was as shown in the last slides. No smooth muscle was found in either one. Recently at The Veterans Hospital one was removed by Dr. Wylie, and it is the only one I know where a connection with the pericardium was found. It was a typical cyst filled with the same kind of fluid and the opening in the pericardium was pre-existent, because it was seen communicating with the pericardium by a very small stalk with patent lumen.

DR. DRASH.—We suggested that the term pericardial cysts be dropped, because in none of our cases nor in any that we could definitely identify in the literature was there any communication with the pericardium. I am not sure that Dr. Berry's case belongs in this group. His case sounds as if it might have been a diverticulum of the pericardium. In all of the cases that we operated upon the cyst came away quite freely. It had a very limited blood supply and they were all covered by mediastinal pleura and apparently originated in the mediastinum. We felt that the term mediastinal cysts should be used rather than the term pericardial cysts. All of these tumors were against the anterior chest wall, but showed no connection with it.

midscapular line to the costochondral junction is useful for pneumonectomy, resection of the upper lobe, and pulmonary decortication. If the incision is carried more anteriorly, the internal mammary artery is exposed to avoid accidental injury to it. Apical segmental resection is best performed through the third or fourth interspace.

The seventh or eighth interspace is satisfactory for vagotomy and the eighth is used for such procedures as repair of diaphragmatic hernia and trans-

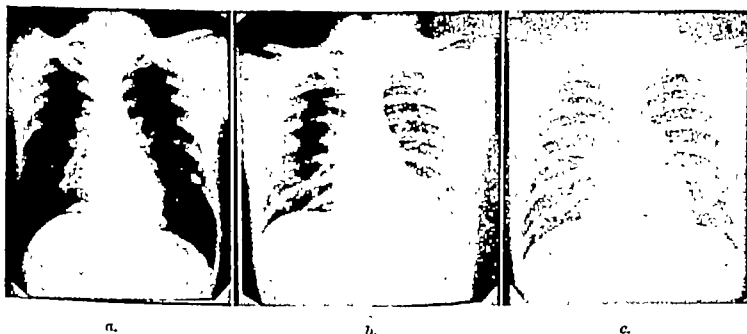


Fig. 2.—a, Preoperative chest roentgenogram. b, First day postoperative, repair of diaphragmatic hernia, showing obscuration of left costophrenic angle. c, One week postoperative, shows complete clearing of the left pleural cavity.



Fig. 3.—a, Preoperative chest roentgenogram. b, Chest roentgenogram two days postoperative, transsternal vagotomy. No evidence of pleural effusion.

thorax and limitation of excursion of the thoracic wall, thereby encouraging retention of pulmonary secretions with its resultant atelectasis and pneumonitis. Grimson referred to a form of bronchitis associated with dyspnea produced by accumulation of tenacious mucus in the trachea in fifteen cases of his series. This seemed to be associated with pain and splinting of the thoracic wall and interference with coughing.

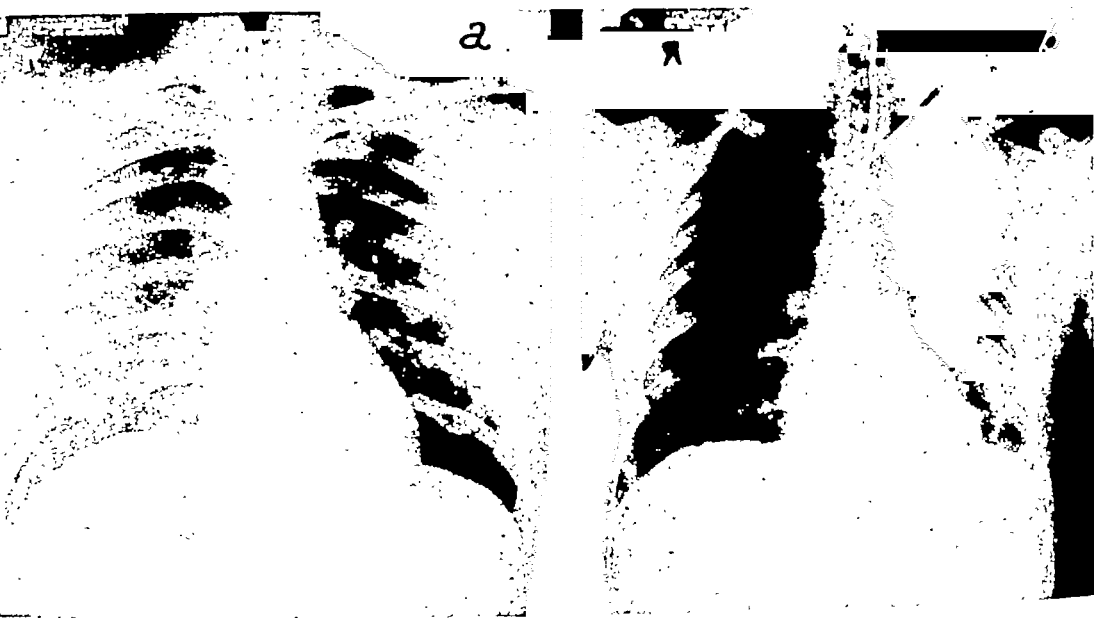


Fig. 1.—*a*, Preoperative chest roentgenogram. *b*, Chest roentgenogram two days postoperative, transthoracic vagotomy, showing slight obscuration of left costophrenic angle.

It is possible in most cases of intercostal incision to obtain accurate closure of the pleura, an accomplishment which is practically impossible with rib resection. The practice of approximating the ribs above and below the resected rib by encircling sutures, which is frequently resorted to, is surgically unsound because of the pressure of these sutures on the intercostal nerves and vessels and on the bone itself. There is good reason to believe that inaccurate closure of the pleura is conducive to pleural effusion in the closed thorax. The fluid of trauma and inflammation, which is a necessary consequence of a long thoracic wall incision, is drawn from the thoracic wall into the pleural cavity by the pumplike action of the lungs in respiration, if a pathway for the escape of the fluid exists.³ With tight closure of the pleura, the escape of the fluid into the free pleural cavity is definitely minimized as shown by follow-up x-ray studies in such procedures as thoracic vagotomy and repair of diaphragmatic hernia (Figs. 1, 2, and 3).

In four patients who had exploratory thoracotomy for nonresectable carcinoma of the lung who have come to autopsy some months later, there was accurate healing with almost imperceptible pleural scar.

The location of the intercostal incision depends upon the operation to be performed. An incision in the fourth or fifth interspace extending from the

such instances exposure is improved by dividing the rib above or below the incision through its costal cartilage. Anterior division is preferable to the posterior because of less irritation to the intercostal nerve. One centimeter of cartilage is resected to lessen the discomfort occasioned by friction of the divided segments.



Fig. 5.—Thorax opened with incision in intercostal space for decortication of lung. Distance between blades of rib spreader (Finoclietto) measures 15 cm.

TECHNIQUE OF OPERATION

The patient is placed in a lateral or semisupine position and an incision from 10 to 14 cm. in length is made through the skin in the selected intercostal space. The overlying extracostal muscles are divided with scalpel or by blunt dissection in the direction of their fibers. The intercostal muscles and parietal pleura are divided with a single incision exactly midway between and parallel to the ribs above and below, thereby avoiding the intercostal vessels and nerve (Fig. 4). A heavy nonyielding rib spreader (we have found the Finoclietto instrument to be ideal) is then inserted between the ribs over gauze pads which serve to protect the intercostal structures from excessive pressure of the spreader. The rib spreader is placed away from the anterior aspect of the incision in order to lessen the tendency of the costal cartilage to become dislocated from the sternum. While this has caused no difficulty

diaphragmatic operations upon viscera in the left upper quadrant of the abdomen.

In our only case of suppurative pericarditis, in which the pericardial sac was opened into the pleural cavity, the seventh interspace did not give satisfactory exposure because of a relatively high adherent diaphragm, and the seventh rib was therefore divided anteriorly.

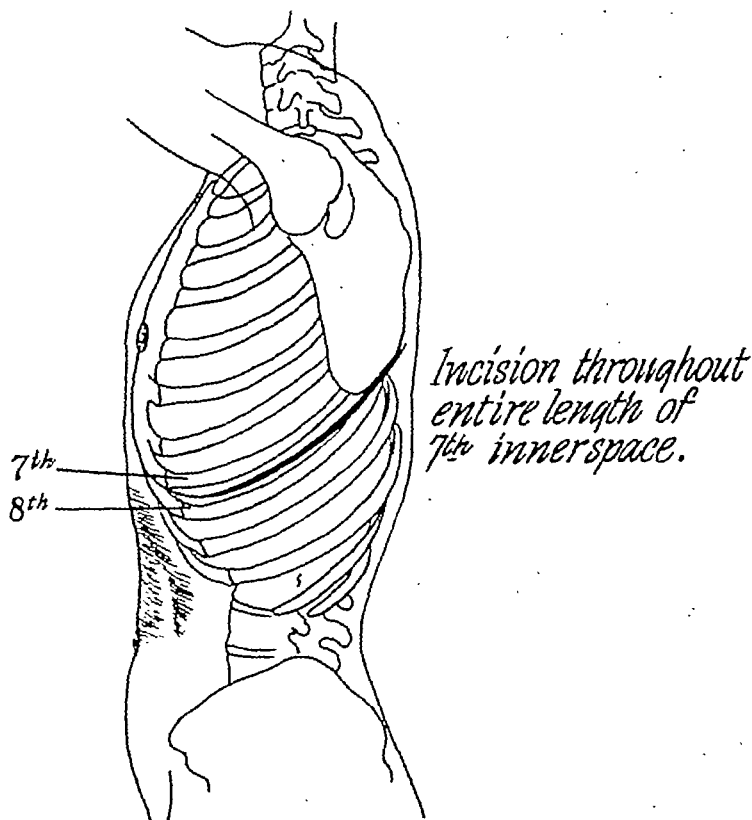


Fig. 4.—Incision is made midway between two ribs in the selected intercostal space.

The seventh interspace is generally the most satisfactory for long incisions. Difficulties with blood vessels are more likely to occur in the region of the interdigitations of the serratus anterior and external oblique muscles in the eighth and ninth interspaces. However, this should in no way deter one from using either the eighth or ninth interspace when indicated. The lower part of the thorax between the seventh and ninth ribs is relatively free of thick muscle tissue except posteriorly, where transection of a relatively small portion of the latissimus dorsi muscle suffices for adequate exposure. The thinner muscles, such as the serratus anterior and the external oblique, are split in the direction of their fibers. As one goes cephalad and posteriorly, more extracostal muscle tissue is encountered, and this together with the scapula increases the amount of dissection. Therefore, when the approach is elective, the seventh or eighth interspace is preferable.

There will be occasions in which one fails to select the proper interspace for the most advantageous exposure, as occurred in three of our early cases. In

diaphragmatic operations upon viscera in the left upper quadrant of the abdomen.

In our only case of suppurative pericarditis, in which the pericardial sac was opened into the pleural cavity, the seventh interspace did not give satisfactory exposure because of a relatively high adherent diaphragm, and the seventh rib was therefore divided anteriorly.

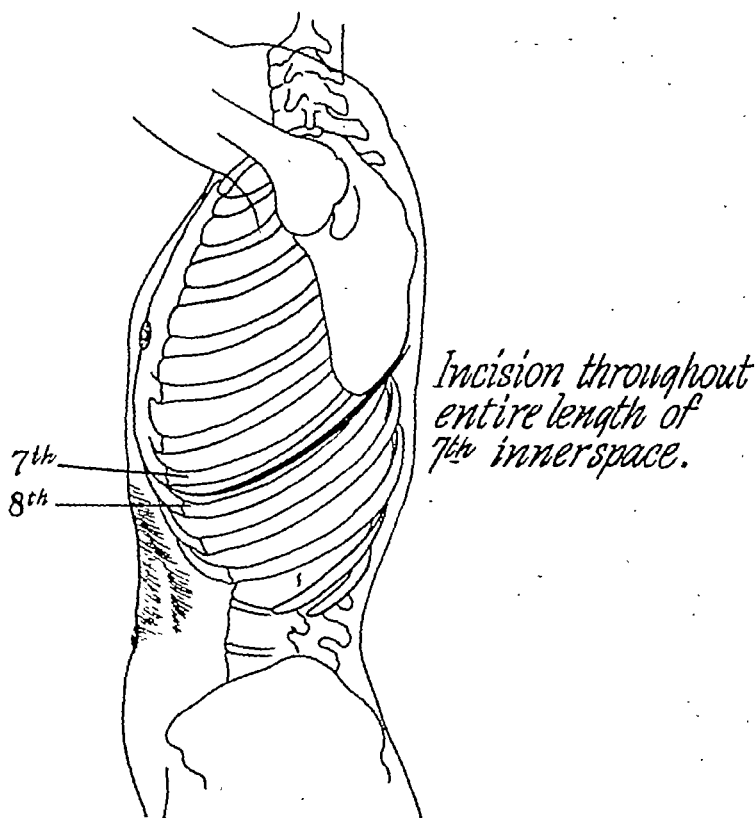


Fig. 4.—Incision is made midway between two ribs in the selected intercostal space.

The seventh interspace is generally the most satisfactory for long incisions. Difficulties with blood vessels are more likely to occur in the region of the interdigitations of the serratus anterior and external oblique muscles in the eighth and ninth interspaces. However, this should in no way deter one from using either the eighth or ninth interspace when indicated. The lower part of the thorax between the seventh and ninth ribs is relatively free of thick muscle tissue except posteriorly, where transection of a relatively small portion of the latissimus dorsi muscle suffices for adequate exposure. The thinner muscles, such as the serratus anterior and the external oblique, are split in the direction of their fibers. As one goes cephalad and posteriorly, more extracostal muscle tissue is encountered, and this together with the scapula increases the amount of dissection. Therefore, when the approach is elective, the seventh or eighth interspace is preferable.

There will be occasions in which one fails to select the proper interspace for the most advantageous exposure, as occurred in three of our early cases. In

According to Thompson,² Freedlander and Gebauer suggested that pericardial defects might be produced by failure of fusion of the embryonic transverse septum with the pleuropericardial or pleuroperitoneal membranes.

Laipply³ is in accord with Lambert¹ in reference to the origin of pericardial cysts.

INCIDENCE

These lesions are not common and are usually found on routine x-ray examinations of the chest. Out of a large series of cysts and cystic tumors of the mediastinum Laipply³ found only four pericardial celomic cysts. Blades,⁴ in a series of 109 cases of mediastinal tumors, found ten pericardial celomic cysts. The higher incidence of these cysts in Blades' series is probably a result of the large number of routine roentgenograms of army personnel. All of the pericardial cysts in his series were asymptomatic. Lam,⁵ in 1947, and others have reported single cases of pericardial celomic cysts.

PATHOLOGY

Pathologically the reported cases of pericardial cysts are similar to the case herein reported. The walls of the cysts are made up of fibrous connective tissue lined by a layer of flattened endothelial or mesothelial cells. Blades⁴ is of the opinion that in the past some of these cysts may have been classified as cystic hygromas or cystic lymphangiomas.

Pericardial celomic cysts are found in the anterior mediastinum in contact with the anterior thoracic wall, the parietal pericardium, and sometimes the diaphragm and the lung.

SYMPTOMS

Pericardial cysts rarely cause symptoms. Pickhardt⁶ reported a case of pleurodiaphragmatic cyst in 1934. This patient was a 53-year-old woman who complained of sharp, knifelike pain over the precordium for a period of several weeks. The case herein reported and Pickhardt's case are the only two in the literature in which symptoms were attributed to a pericardial cyst. Both patients were relieved by removal of the cyst.

DIAGNOSIS

In an attempt to establish the diagnosis of pericardial cyst, bronchiogenic carcinoma, diaphragmatic hernia through the space of Morgagni, and dermoid tumor are the lesions most likely to be confused with benign cyst.

The history of cough is suggestive of bronchiogenic carcinoma. Careful roentgenologic studies with instillation of Lipiodol in the bronchial tree, bronchoscopy, and histologic study of cells aspirated through the bronchoscope will aid in making a positive diagnosis of carcinoma.

Herniation of omentum through the space of Morgagni can be confusing in the differential diagnosis. An upper gastrointestinal series should be done in all of these patients in order to rule out the presence of herniated stomach or

Pathologic Report.—Gross description: The pericardial cyst consisted of a clear wall multilocular cyst measuring 8 cm. in diameter. The walls were thin and the blood vessels prominent. There was a small amount of fat around the periphery.

Microscopic description: Section through the wall of the cystic cavity showed the lining to consist of endothelial cells which in some areas were elongated and flattened. In others they were two to three cell layers deep and here assumed a more cuboidal appearance. Their nuclei were round to oval, deeply stained, and adult in age. The amount of cytoplasm was moderate with light pink staining characteristics. The underlying tissue consisted of edematous connective tissue and varying amounts of fat. The small capillaries were adult in age and congested. The cyst cavity was irregular in outline and showed small protrusions from the surface into the lumen of the cavity forming small papillary projections. These consisted of a stalk of fat and fibrous tissue covered with the same type of endothelial cells as described for the major portion of the cyst.

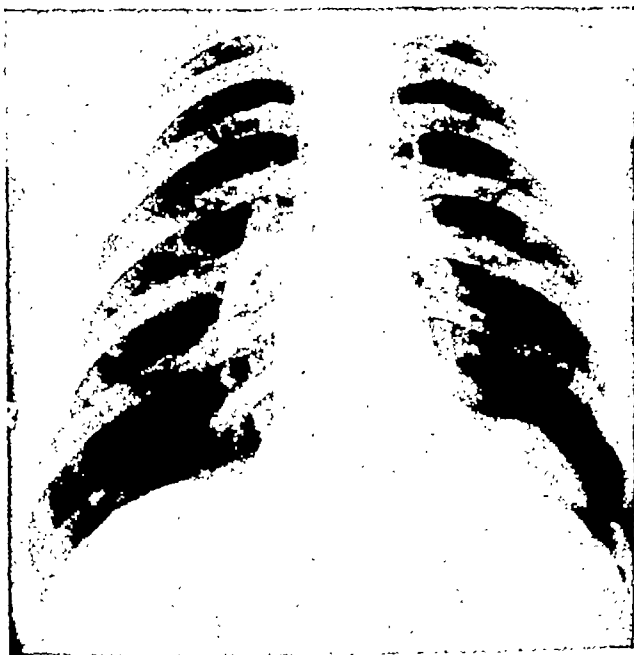


Fig. 1.—Preoperative roentgenogram of chest.

DISCUSSION

Pericardial cysts are usually discovered on routine x-ray examination of the chest. The patient reported here was disturbed by an uncomfortable feeling in the left chest and consulted his family physician.

PATHOLOGY

Lambert¹ attributed the origin of pericardial cysts to failure of primitive mesenchymal lacunae which form the pericardium to fuse with others and instead form independent cavities. Failure of fusion of these lacunae may result in other anomalies such as congenital absence of the pericardium, congenital diverticulum of the pericardium, or congenital hernia of the diaphragm.

Lambert¹ proposed the name, pericardial celomic cysts, as being a more accurate term to define their true character. Lambert¹ also reported two cases of pericardial celomic cyst in his paper.

CASE HISTORY

The patient, a 57-year-old, well-developed, well-nourished man fell through a manhole in September, 1948, injuring the right thorax, with subsequent sharp pains in the right chest and upper abdomen and slight cough. Roentgenograms of the chest at the time were negative, but he continued to have vague pains. He was admitted to the hospital on Feb. 1, 1949, and presented fluid in the right chest. General condition was excellent, though for the previous three weeks there had been marked loss of appetite. Bronchoscopy showed compression of

Fig. 4.

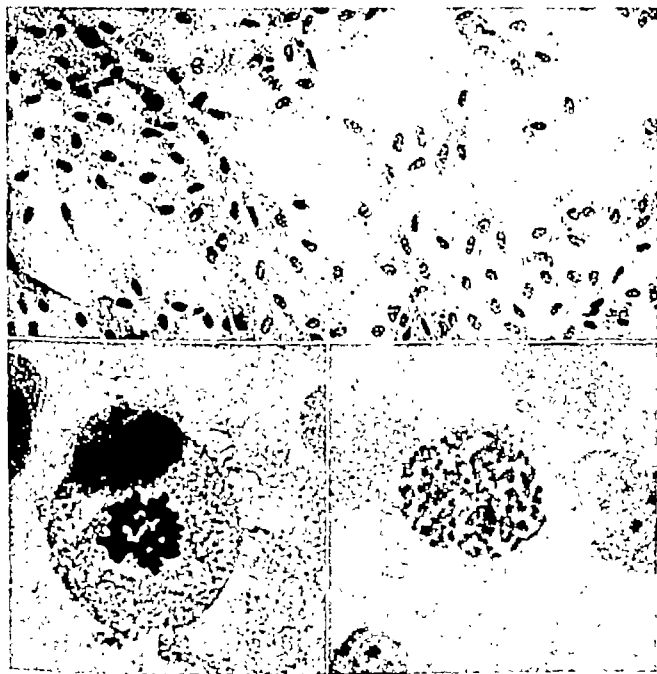


Fig. 5.

Fig. 6

Fig. 4.—The tendency to alveolar arrangement in tissue culture is here demonstrated. Stain Crt 304 ($\times 200$).

Fig. 5.—Fairly well-balanced mitosis in tissue culture. Stain CR 304 ($\times 1400$).

Fig. 6.—Multipolar mitosis in tissue culture. CR 304 ($\times 1400$).

the right main bronchus, but there were no mucosal lesions. An exploratory operation was done,* but the pleura was completely involved, as was the pericardium. Careful examination and multiple sections at post-mortem† revealed this tumor to be grossly and microscopically a mesothelioma with none of the epithelial-like elements present. No tumors were located elsewhere, notwithstanding multiple sections. Was this, therefore, originally a well-localized operable tumor as its histology would suggest, or a diffuse type, as one would surmise from the clinical history?

*By Dr. W. E. Burnett and Dr. G. Rosemond.

†By Dr. Paul S. Guerin, Resident in the Department of Pathology.

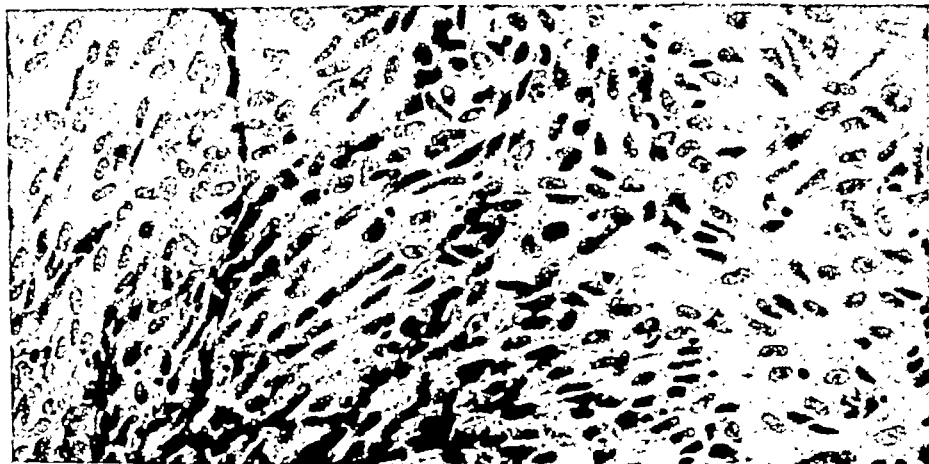


Fig. 1.

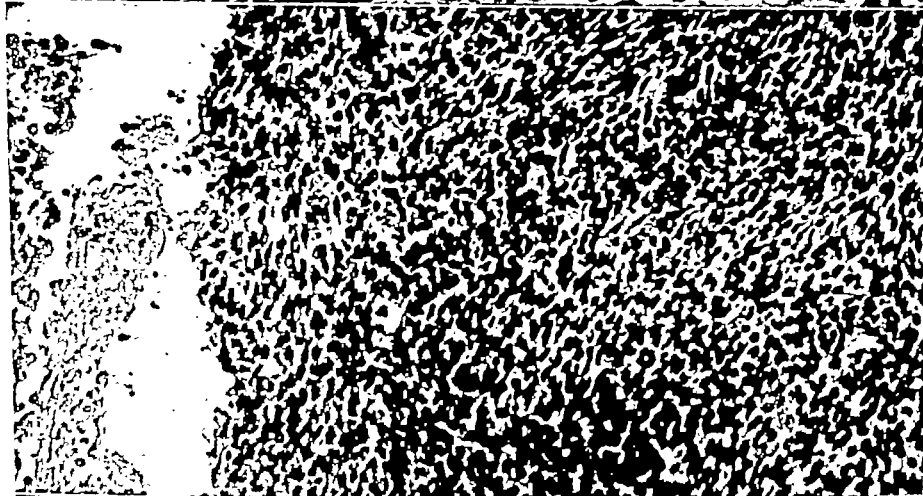


Fig. 2.

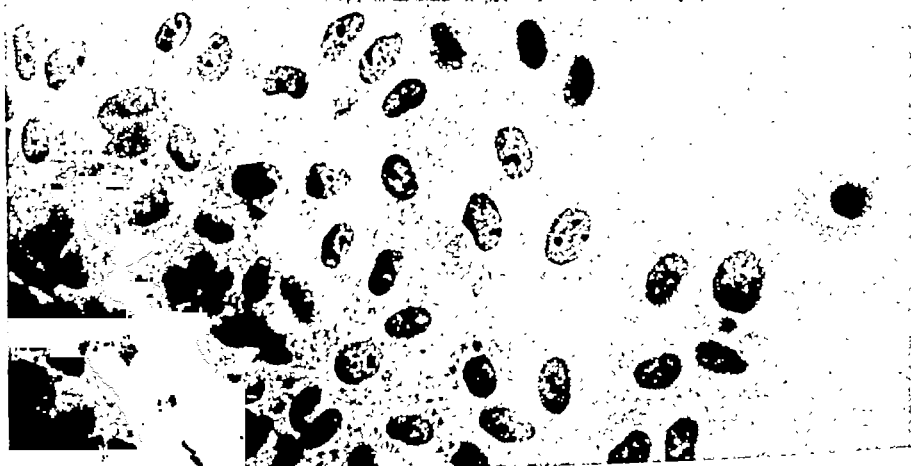


Fig. 3.

Fig. 1.—Tissue culture showing the spindling of the cells. Stain CR 304, (18) ($\times 200$).

Fig. 2.—Section of the tumor. Stain CR 304 ($\times 200$).

Fig. 3.—Note the flattened cells growing out in sheets in tissue culture. Stain CR 304 ($\times 400$).

Where growth was unusually rapid, the cytoplasm was extremely light and delicate. The intercellular boundaries were usually well marked. The cells had a plump oval nucleus with fine to medium heavy metachromatic threads and two or more nucleoli.

In some of the smaller fragments the cells grew out in sheets at first, then while still flattened (in form) they tended to form alveoli which were not secondary to fibrinolysis (Fig. 4). There was in fact, very little liquefaction even over a period of eighteen days. Syneresis was marked in the human plasma, but that is a common occurrence within the first twelve hours.

The mitoses in the cells were as often balanced as unbalanced. In the unbalanced form there was remarkably little change in the shape of cells (Figs. 5 and 6). Normal wandering cells were few and rare in both media, the growth being formed mainly by tumor cells.

The growth in the chicken plasma was different. Some of the cells grew in sheets (Fig. 7), but as the new growth became about three cells deep, the cells took on a rounder form with the nuclei placed near the periphery. These cells isolated themselves. Their outline was irregular and they had a tendency to assume bizarre forms (Fig. 8) with numerous atypical mitoses. These cells did not resemble any of the cells seen in the original tumor masses, but were very similar in character to the epithelial-like cells seen in the usually rapidly spreading tumor of the pleura. None of these cells were found in any of the multiple sections taken of the tumor post mortem.

Once again we observed, therefore, as one of us¹² has noted on numerous occasions in the study of human tumors and lymph nodes for the past ten years, that the form a tumor assumes in the host is in part, at least, dependent on a factor present in the host, besides the purely physical environmental factors. This elusive factor can usually be demonstrated in the plasma of the host. Many workers have observed the difference between human plasma of cancer patients and of normal individuals. There is a difference in the coagulation,¹³ frequently a difference in pH reading,^{14, 15} a difference in the plasma pattern,¹⁶ and in its reducing power.¹⁷ Some of these observations have been verified, others have been contradicted, yet, there is some truth in most of them, though none of them as yet stand up as reliable cancer tests. Whether this is one and the same factor or a different one has not been verified because the observations under present techniques are still too inconstant.

DISCUSSION

In the pleural tumor under consideration only the spindle form of cell was apparent, but under the different conditions created by tissue culture, the epithelial-like form was demonstrated as well. Clinically, moreover, the diffuse and rapid growth resembles the behavior of the mixed form of pleural mesothelioma, further emphasizing the importance of the tissue culture findings.

One of the determinants of the dominant cell types is evidently present in part in the plasma of the tumor host. Whether it is a reaction of the host to the tumor or whether it is a physicochemical property in the plasma or tissues of the host which precedes tumor development, we were unable to demonstrate. We do know from experience that when tissue culture is used as a diagnostic aid in atypical tumors, it is always advisable to use the autogenous plasma, in addition to the standard chicken plasma. Actual and potential forms of tumors can thus not infrequently be demonstrated.

In reviewing the literature we have been impressed by the resemblance of the tissue culture of this tumor with that of the pleural mesothelioma studied by Stout and Murray. It further emphasizes the value of this method in the study of unusual tumors and the consistency of growth in tissue cultures of similar tumors.

Behavior of the Tumor in Tissue Culture.—Fragments of necrotizing tumor and firm tumor nodules were taken at the time of operation. These were inoculated into two types of media: Tyrode's solution, 1 part; mouse embryo extract, 1 part; and autogenous human plasma, 1 part. In the other medium, chicken plasma was substituted for human plasma.^{11, 12}

Fig. 7.

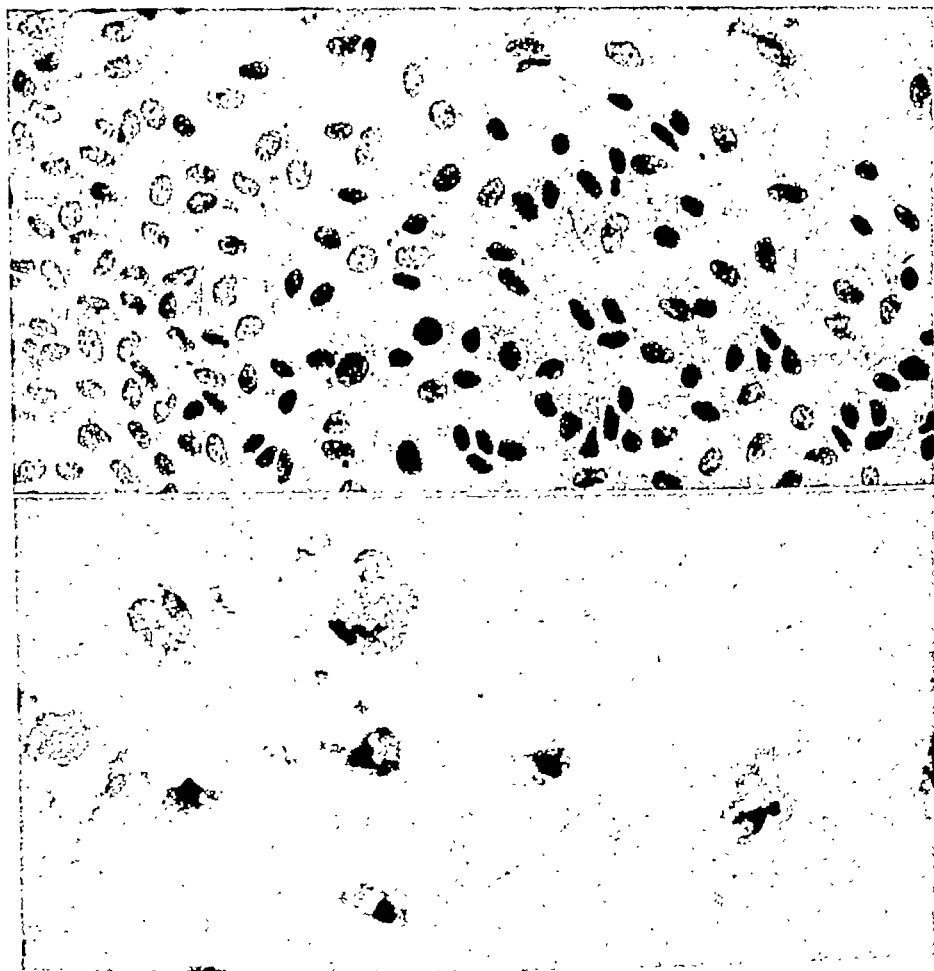


Fig. 8.

Fig. 7.—Tissue culture of tumor in medium containing chicken plasma. Stain CR 304 ($\times 200$).

Fig. 8.—Bizarre forms of isolated cells near periphery of growth in tissue culture in chicken plasma medium. Stain CR 304 ($\times 1400$).

There were two outstanding characteristics common to the growth in either media. One was the rapidity of growth which was profuse within twenty-four hours. The other was that, from minute particles of seemingly nonviable tissues containing three to four picnotic nuclei, tumor cells proliferated within forty-eight hours, reproducing rapidly. In the human plasma the fixed cells growing out from the thicker fragments were at first more spindle shaped (Fig. 1), similar in appearance to the arrangement in the section of the tumor (Fig. 2). As the cells became less crowded, they flattened out, growing in membranes. They had a mosaic arrangement (Fig. 3), which was very characteristic and emphasized by phase illumination. Under this light, an accumulation of fine mitochondria in the cytoplasm and surrounding the nucleus could be seen. The outlines of the unstained cells were almost invisible unless examined under reflected light. The layers closest to the glass coverslip grew most rapidly.

ARTERIOVENOUS FISTULA OF THE LUNG

WITH REPORT OF A CASE

JAMES CARSWELL, JR., M.D.*

McKINNEY, TEXAS

SINCE 1942, when Hephurn and Dauphinee⁸ reported a case of hemangioma of the lung diagnosed clinically and successfully treated by pneumonectomy, an increasing number of reports of pulmonary arteriovenous fistula has appeared in the literature. These authors reviewed the literature prior to their paper and found few references describing the occurrence of this lesion. Jones,¹⁰ in 1944, reported the first case of multiple cavernous hemangioma of the lungs successfully treated by local resection of the tumors. Since the lesions were bilateral, treatment was carried out in two stages, approximately eight months apart. Maier and associates,¹³ in 1948, presented a study of the circulatory dynamics in pulmonary arteriovenous fistula and included a case of their own complicated by bacterial endarteritis and successfully treated by chemotherapy and lobectomy. In the discussion of Maier's paper, several other cases of arteriovenous fistula of the lung were brought to light. Wodehouse,¹⁵ in 1924, added two cases previously unreported, one of which was complicated by a brain abscess. Additional reports^{1, 2, 3, 5, 7, 8, 11, 14, 16, 17} bring the total number of cases in the literature to twenty-eight.

It is likely, as Wodehouse implied, that the earlier reports have resulted in an increased awareness of arteriovenous fistula of the lung as a clinical entity. The signs and symptoms, although variable¹³ should arouse suspicion of such a lesion. Those most frequently mentioned in the reported cases were cyanosis, polycythemia, clubbing of the extremities, and the appearance of a pulmonary density by x-ray examination. An audible murmur over the lesion and expansile pulsation of the mass fluoroscopically are inconstant findings. Dyspnea,^{9, 12} hemoptysis,^{6, 9} and the occurrence of coexisting hemangiomas elsewhere in the body may appear as prominent symptoms in some cases of arteriovenous fistula.^{4, 13, 15}

CASE REPORT

Illness at Time of Admission.—A 20-year-old delivery-truck driver was admitted to this hospital February 23, 1948, because of recurrent mild, shooting pains in the inter-scapular region, of two years' duration. The pain usually lasted several minutes, recurring at irregular intervals of a few days to weeks, and was not incapacitating. It was unrelated to exercise, emotional excitement, or the ingestion of food, and was not initiated or aggravated by deep breathing. There had been no cough, dyspnea, hemoptysis, weight loss, or loss of appetite. In April, 1946, one month after the onset, a chest roentgenogram revealed a small, circumscribed area of increased density in the posterolateral aspect of the right lower lobe. In May, 1946, a bronchogram was done but contributed no further information. Bronchoscopy in November, 1947, was negative. The patient was referred to this hospital for further study and exploratory thoracotomy.

Sponsored by the Veterans Administration and published with the approval of the Chief Medical Director. The statements and conclusions published by the author are the result of his own study and do not necessarily reflect the opinion or policy of the Veterans Administration.

Received for publication June 9, 1949.

*Chief of Thoracic Surgery Section, Veterans Administration Hospital.

RÉSUMÉ AND CONCLUSIONS

1. Report of a case of pleural mesothelioma with rapid diffuse spread is presented.
2. The histopathologic characteristics seen are those of the fibrosarcomatous slow-growing type, and therefore operable.
3. Tissue culture in autogenous plasma reveals the mesothelial character of the cells.
4. Tissue culture in chicken plasma reveals the potential epithelial-like cell form, placing it in the rapidly spreading group, usually inoperable.
5. Discussion of the factors influencing the form of the mesothelial cell is presented.

Note.—We are indebted to Carol Bocher, B.S., for valuable technical assistance.

REFERENCES

1. Stout, A. P., and Murray, Margaret, R.: Localized Pleural Mesothelioma. Investigation of Its Characteristics and Histogenesis by the Method of Tissue Culture, *Arch. Path.* 34: 951-964, 1942.
2. Robertson, H. E.: Endothelioma of the Pleura, *J. Cancer Research* 8: 317-375, 1924.
3. Willis, R. A.: Metastatic Deposit of Bronchial Carcinoma in Hydrocele Misdiagnosed "Endothelioma" With Review of Supposed "Endothelioma" of Serous Membranes, *J. Path. & Bact.* 47: 35-42, 1938.
4. Lewis, W. H.: Mesenchyme and Mesothelium, *J. Exper. Med.* 38: 257-261, 1923.
5. Holtfreter, J.: Concepts on the Mechanism of Embryonic Induction and Its Relation to Pathogenesis and Malignancy, *Symposia of the Society of Experimental Biology* No. 2, New York, 1948, Academic Press, Inc., pp. 17-46.
6. Young, J. S.: The Experimental Production of Metaplasia and Hyperplasia in the Serosal Endothelium, and of Hyperplasia in the Alveolar Epithelium of the Lungs of the Rabbit, *J. Path. & Bact.* 31: 265-275, 1928.
7. Fischer, B.: Ueber Experimentelle Erzeugung Grosser Flimmer Epithelblasen der Lunge, *Frankfurt. Ztschr. f. Path.* 27: 98-184, 1922.
8. Fischer, B.: Ueber Experimentelle Erzeugung von Epithelwucherungen und Epithel-metaplasie, *Verhandl. d. deutsch. path. Gesellsch.* 10: 20, 1906.
9. Maximow, A. A.: A Textbook of Histology, Philadelphia, 1948, W. B. Saunders Company, pp. 38.
10. Hochberg, L. A., Epstein, I. G., and Pernikoff, M.: Endothelioma (Mesothelioma) of Pleura, *Dis. Chest* 13: 621-626, 1947.
11. Sano, M. E., and Smith, L. D.: Tissue Culture as a Diagnostic Aid in the Identification of Atypical Tumors, *Arch. Path.* 30: 504-508, 1940.
12. Sano, M. E.: Cancer. (In press.)
13. Huggins, C., Miller, M. G., and Jensen, E. V.: Thermal Coagulation of Serum Proteins. II. Deficient Coagulation in Cancer and the Iodoacetate Index, *Cancer Research* 9: 177-182, 1949.
14. Slosse, A.: See Reding.¹⁵
15. Reding, R.: *Le terrain cancéreux et cancérisable*, Paris, 1932, Masson & Cie.
16. Sano, M. E.: Unpublished personal observations.
17. Weiss, Emil: Color Reactions With Malignant Sera. Paper Given at the 40th Annual Meeting of The American Association for Cancer Research, April 16, 1949.
18. Sano, M. E.: Trichome Stain for Tissue Section, Culture or Smear, *Am. J. Clin. Path.* 19: 898, 1949.
19. Gunn, Frances D.: In Anderson, W. A. D.: *Pathology*, St. Louis, 1948, The C. V. Mosby Company, pp. 748.

past six years, however, twenty-eight cases have been reported and it is probable that others have been observed.

2. A case of arteriovenous fistula of the lung is reported, in a patient whose complaint of mild interseapular pains prompted the taking of a chest film.

3. The diagnosis of pulmonary arteriovenous fistula may be made without difficulty in those patients presenting themselves with one or more of a group of symptoms commonly attending this disease. In other instances the presence of a vascular lesion in the lung may remain unsuspected for years.

REFERENCES

1. Abbott, Osler: Discussion of Maier et al., *J. THORACIC SURG.* 17: 25, 1948.
2. Adams, W. E., Thornton, T. F., and Eichelburger, L.: *Arch. Surg.* 49: 5, 1944.
3. Adams, W. E.: Discussion of Jones and Thompson, *J. THORACIC SURG.* 13: 357-371, 1944.
4. Blagard, Dewey: Discussion of Maier et al., *J. THORACIC SURG.* 17: 25, 1948.
5. Boerema, I., and Brilman, R. P.: *J. THORACIC SURG.* 17: 705-708, 1948.
6. Bowers, W. F.: *Nebraska Med. J.* 21: 55, 1936.
7. Byron, Francis R. X.: Discussion of Maier et al., *J. THORACIC SURG.* 17: 25, 1948.
8. Goldman, Alfred: *J. Lab. & Clin. Med.* 32: 330, 1947.
9. Hepburn, J., and Dauphinee, J. A.: *Am. J. M. Sc.* 204: 681, 1912.
10. Jones, R. M.: *Brit. J. Surg.* 31: 270, 1944.
11. Jones, R. M.: Discussion of Jones and Thompson, *J. THORACIC SURG.* 13: 357-371, 1944.
12. Jones, John C., and Thompson, W. Paul: *J. THORACIC SURG.* 13: 357-371, 1944.
13. Maier, Herbert C., Himmelstein, A., Riley, Richard L., and Bunin, Joseph J.: *J. THORACIC SURG.* 17: 13-26, 1948.
14. Samson, Paul C.: Discussion of Maier et al., *J. THORACIC SURG.* 17: 23, 1948.
15. Shells, Lawrence M.: Discussion of Maier et al., *J. THORACIC SURG.* 17: 23, 1948.
16. Sweet, Richard H.: Discussion of Maier et al., *J. THORACIC SURG.* 17: 23, 1948.
17. Watson, W. Law: *Surgery* 22: 909-929, 1917.
18. Wodehouse, G. E.: *J. THORACIC SURG.* 17: 408-415, 1948.

gave no additional information. The patient produced no sputum, so bacteriologic studies were not done. The appearance and behavior of the lesion in the lungs, mild polycythemia, and clubbing of the fingers suggested the diagnosis of a vascular tumor, and it was decided to explore the chest.

Operation.—Thoracotomy was performed March 9 and the pleural space was found to be free of adhesions. There was a visible pulsating mass about 4 cm. in diameter immediately beneath the pleura in the inferior posterolateral aspect of the lower lobe. The thinness and transparency of the pleural covering made it possible to observe the blood eddying within the tumor. The upper and middle lobes were normal to inspection and palpation. The lower lobe was removed without incident.

Subsequent Course.—The patient's convalescence was uneventful and he was discharged from the hospital on the fourteenth postoperative day.

Gross Pathology.—The specimen consisted of the right lower lobe of the lung. On the posterolateral aspect near the base there was a soft cystlike area measuring 2.5 cm. in diameter and covered with a thin, somewhat translucent pleura. The remainder of the lobe presented no gross pathologic changes. A cannula was introduced into the bronchus and the lobe inflated, after which the bronchus was tied. A second cannula was tied into the main artery and injected with radiopaque material after infusing with saline solution to remove the blood. These solutions returned freely through the open stump of the vein. Roentgenograms of the inflated, injected lobe were made in three views before dissection of the specimen was begun. The view taken from above, with the lobe resting with its base on the cassette, shows the lesion most distinctly (Fig. 3). Dissection of the fistula along with its main arterial and venous connections was carried out, and a sketch of the dissection drawn (Fig. 4). The sac itself was unilocular, thin-walled, and lined with endothelium. It contained congealed radiopaque material admixed with blood elements. There were numerous small communications, 1.0 mm. or less in diameter, opening into the lumen of the sac. The major vessels communicating with the sac were themselves grossly normal in appearance and were not appreciably dilated. The bronchial artery was not found. The total diameter of the direct, measurable arterial communication with the sac was 12 mm., while that of the venous side was 10 mm. Numerous smaller communicating channels radiated from the wall of the sac to be lost in the surrounding parenchyma. No connection with a bronchial vessel could be demonstrated.

COMMENT

The case presented here illustrates further the variety of symptoms which may be encountered in patients with arteriovenous fistula of the lung. Mild, shooting pain in the interseapular region, intermittent and transient in character, was the only symptom of which the patient complained. Very slight polycythemia and minimal clubbing of the fingers were present. Prior to admission to the hospital, chest roentgenograms were taken and the patient was suspected of having a tuberculoma, although the possibility that the lesion might be a vascular tumor was entertained. The preoperative diagnosis was of possible vascular tumor of the right lower lobe. Unfortunately, a hematocrit was not obtained prior to surgery for later comparison. Seven weeks after operation the red blood count was 5.5, hemoglobin 16.5, a slight decrease as compared with the preoperative determinations. Three days after operation the hematocrit was 53 as compared with 49 seven weeks later.

SUMMARY

1. Prior to 1942, few references to pulmonary arteriovenous fistula were found in the literature, and the disease was considered extremely rare. In the



Fig. 1.



Fig. 2.

Fig. 1.—Posteroanterior roentgenogram (July 10, 1944) reveals a sharply circumscribed tumor mass in the region of the upper pole of the left hilum measuring approximately 7 cm. in diameter and obstructive emphysema of the left upper lobe.

Fig. 2.—Lateral roentgenogram reveals mass in posterior mediastinum.

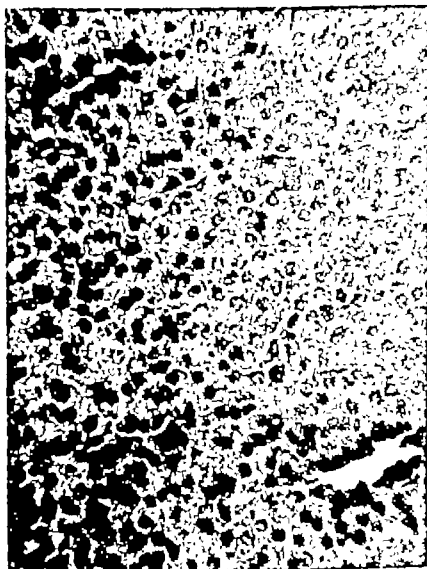


Fig. 3.—High-power field of tumor shows dense array of plasma cells with typical eccentric nuclei. (Magnified.)

PLASMA CELL TUMORS OF THE MEDIASTINUM AND LUNG

REPORT OF TWO CASES

WILLIAM G. CHILDRESS, M.D., VALHALLA, N. Y., AND
GEORGE C. ADIE, M.D., NEW ROCHELLE, N. Y.

IN 1943 in a comprehensive review of the literature Hellwig¹ collected 127 cases of extramedullary plasma cell tumor, and reported a single one of his own. Gordon and Walker² reported a single case of plasmacytoma of the lung in 1944. They were unable at that time to find an instance of plasmacytoma of the lung such as they reported in the literature. However, they stated that Dr. F. W. Stewart had observed two instances of solitary plasmacytoma of the lung. Bross³ found a plasma cell tumor of the mediastinum in a 54-year-old woman at necropsy, and Klose⁴ observed a similar tumor of the pleura in a 61-year-old man. We have been unable to find additional reports in the literature of either pulmonary or mediastinal tumors of this type. The two following cases were found to have plasma cell tumors in these uncommon locations.

Differences of opinion have been expressed regarding the nature of these tumors. Some regard them as carcinoma, others as granulomatous lesions resulting from chronic inflammation, and others consider them benign tumors. The tumor is characterized by an abundance of plasma cells, arranged in palisades or cartwheel distribution, and varying in size and shape with a large amount of basophilic cytoplasm. The cells often contain two or more nuclei, and giant cells and Russell bodies may be present. The tumors are widely distributed, but are more often found in the upper part of the respiratory tract, in the nose, nasopharynx, mouth, and tonsils. Other sites are stomach, intestines, thyroid gland, kidney, ovary, skin, lymph nodes, and spermatic cord.

CASE 1.—M. R., a 37-year-old white male laundry worker, was first seen (by W. G. C.) June 7, 1944.* He was rejected by the Army on the basis of induction chest roentgenogram. For about six months previously there were times when he was somewhat short of breath, but there were no other symptoms and he had gained twenty pounds during the preceding two years. Past and family history was noncontributory. There was no tuberculosis in the family. Physical examination revealed a well-developed and nourished white man not appearing ill. The lungs were clear to percussion and auscultation. Roentgenograms revealed a sharply circumscribed tumor mass in the posterior mediastinum extending out from the upper pole of the left hilum and measuring about 7 cm. in its greatest diameter. Oral temperature was 99° F., pulse 84, blood pressure 118/80. Laboratory findings were as follows: red blood cells 5.2; hemoglobin 13.5 Gm.; white blood cells 5,850; segmentals 48; lymphocytes 43; mononuclears 8. Sedimentation rate, Westergren method, was 32 mm. in one hour. Urine was negative; blood Wassermann was negative. Sputum negative on concentrate for acid-fast organisms. Bronchoscopic examination elsewhere† was essentially negative except for congestion and edema of the left lower lobe bronchus with some constriction.

The patient was referred to Grasslands Hospital, June 27, 1944, for an exploratory operation. This was done (by G. C. A.) on July 11, 1944, under cyclopropane anesthesia.

From the Division of Diseases of the Chest and the Division of Surgery, Grasslands Hospital, Valhalla, N. Y.

Received for publication June 13, 1949.

*Referred by Dr. R. E. Machan, Mamaroneck, N. Y.

†By Dr. Verne Peterson.

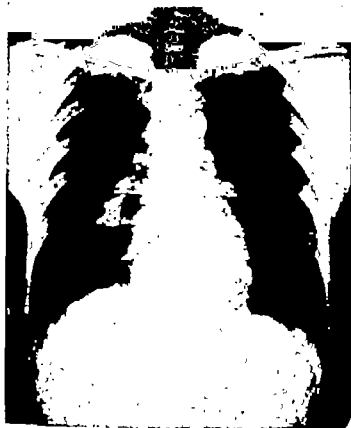


Fig. 5.



Fig. 6.

Fig. 5.—Posteroanterior roentgenogram (April 1, 1948) reveals density between the eighth and ninth posterior ribs on the right.

Fig. 6.—Lateral roentgenogram reveals well-circumscribed mass in region of interlobar septum, involving portions of middle and upper lobes.

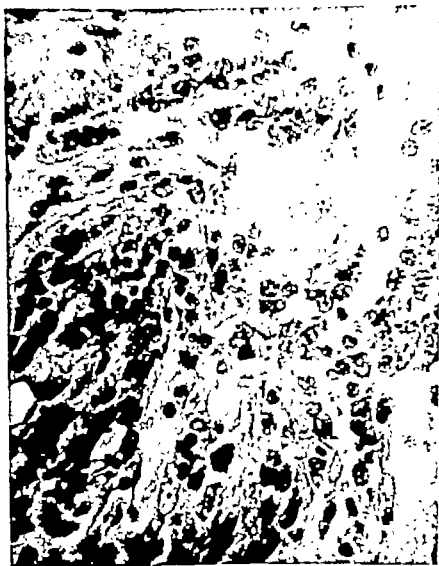


Fig. 7.—High-power field of tumor shows groups of plasma cells in dense fibrous stroma. Lymphocytes and other large mononuclears also present. (Magnified.)

A round firm tumor measuring 2 inches in diameter was observed filling the space formed by the arch of the aorta and apparently attached to the pleura superior to the root of the lung. The pleura was incised over the tumor and by careful, sharp dissection the tumor was removed. Postoperative course was uneventful except that the wound healed rather slowly, and the patient remained in the hospital until Aug. 22, 1944, the forty-second postoperative day.

Pathology: The tumor was spindle shaped measuring 6.5 by 3.5 by 3.5 cm. There was a thin capsule enclosing the mass, with several small friable blood-stained nodes present on the surface. Cut section showed the tissue to be rather dark, dry, and friable similar to blood clot. In fact, there was considerable blood throughout, and at times small irregular blood-filled spaces were encountered. In one area, a pattern of thin lines, radiating at right angles from the capsule, was noted. The sections showed a neoplasm composed of masses of mature, but not always typical, plasma cells growing in a very sparse reticular stroma, highly vascularized. The growth was surrounded by a rather thick collagenous capsule which sent heavy trabecular septa into the tumor. **Diagnosis*** was plasmacytoma.†

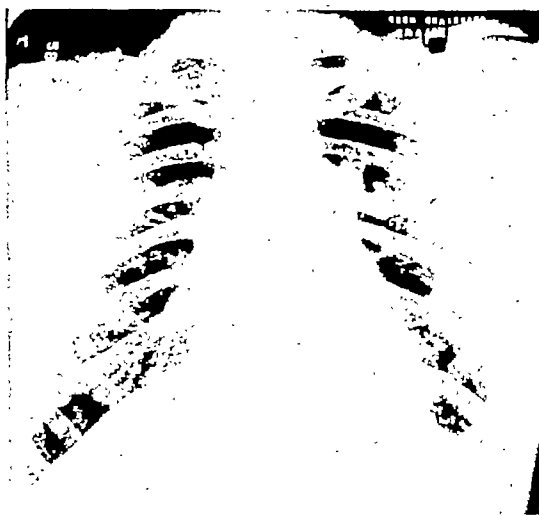


Fig. 4.—Follow-up roentgenogram (Oct. 26, 1948) taken fifty-one months after operation shows no evidence of recurrence.

This patient has been followed by roentgenographic study at intervals since, and except for a virus pulmonary infection in July, 1947, there have been no symptoms referable to the chest, nearly five years after operation. Roentgenogram on Oct. 26, 1948, revealed no evidence of recurrence.

CASE 2.—H. Y., a 21-year-old white man, was first seen (by W. G. C.) on Jan. 30, 1948.† At the time he was employed by a cleaning and dyeing establishment where he claimed to have inhaled formaldehyde fumes. He was separated from the Army in July, 1945, after serving one year, mainly in the Philippines. He had no illness while in the Army, and both induction and separation chest roentgenograms were said to be negative.

He gave a history of a dry hacking cough of about six months duration, and for the previous two months the cough had become productive of about two ounces of yellowish sputum daily. The sputum, on one occasion, was blood-streaked. There were no other symptoms except for a loss of ten pounds during the preceding twelve months. Best weight was 174 pounds, and present weight was 155 pounds. In January he consulted his family physician who obtained chest roentgenograms and a sputum examination, which was found to be negative for acid-fast organisms on direct smear. Subsequent cultures were also negative.

*By Dr. George McClure, Grasslands Hospital pathologist.

†Dr. F. W. Stewart* of Memorial Hospital, New York City, studied the sections and concurred in the diagnosis.

‡Referred by Dr. George L. Daniels of Port Chester, N. Y.

countered. Lymphocytic aggregates were abundantly present, as were many incorporate reticulum cells. No Reed-Sternberg giant cells were seen. Eosinophiles were generously scattered throughout. Remaining alveoli present in the mass were often filled with large foamy macrophages. Microscopically the mass was not so well circumscribed as described grossly. The interlobar fissure and pleura were markedly thickened.*

Plasma cells have been considered to be differentiated lymphocytes, normal constituents of connective tissue originating from tissue lymphocytes, or emigrated blood lymphocytes. Functionally they have been thought to be related to immunization processes, of elaborating antitoxic substances, and capable of removing cellular metabolic products. Plasma cell tumors occur frequently in the bone marrow, but extramedullary plasma cell tumors are more rare and originate more often in the upper respiratory tract. They may be single or multiple, noncancerous or cancerous, with or without metastases. They more frequently metastasize to bones and lymph nodes. Eggston⁶ has observed that plasmaeytomas in the upper respiratory tract become malignant in some instances. He states that an increase in plasma cells may occur in a variety of chronic inflammatory conditions such as tuberculosis and syphilis, to such a degree as to simulate true neoplasm.

In both of our cases symptoms had existed for approximately six months and in Case 1 were undoubtedly due to pressure. However, in Case 2 there were productive cough, blood-streaked sputum, and loss of weight, symptoms not uncommon in pulmonary infection. There has been no evidence of recurrence in Case 1 in more than four years observation, or in Case 2 in ten months observation.

REFERENCES

1. Hellwig, C. A.: *Arch. Path.* 36: 95, 1943.
2. Gordon, Joseph, and Walker, Gerald: *Arch. Path.* 37: 222-224, 1944.
3. Bross, K.: *Folia haemat.* 45: 137, 1931, cited by Hellwig.¹
4. Klose, H.: *Beitr. z. klin. Chir.* 74: 20, 1911.
5. Stewart, F. W.: Personal communication.
6. Eggston, A. A., and Wolff, D.: *Histopathology of the Ear, Nose and Throat*, Baltimore, 1947, Williams & Wilkins Company.

*Dr. Gross, suspecting a plasma cell tumor, consulted Dr. F. W. Stewart, of Memorial Hospital, New York City, who attached the diagnosis of "plasma cell granuloma of lung." He stated that he had seen two or three similar examples and believed that they are not tumors, but represent inflammatory granulomatous lesions of unknown etiology.

Past and family history was noncontributory. There was no tuberculosis in the family. Physical examination revealed a rather pale but well-nourished white man of about the stated age with entirely normal physical signs. The lungs were clear to percussion and auscultation. The chest roentgenogram revealed a dense, fairly well-circumscribed elliptical mass in the area of the right middle lobe measuring approximately 4 cm. in its greatest diameter. Blood pressure was 108/78. Laboratory findings were as follows: red blood cells 4.95; hemoglobin 14.5 Gm.; white blood cells 7,800; segmentals 74; lymphocytes 22; mononuclears 2; eosinophile 1. Sedimentation rate, Westergren method, was 7 mm. in one hour. Blood chemistry was normal. Bence-Jones protein test was negative; nonprotein nitrogen 35; sugar 80. Blood Wassermann was negative. Sputum and gastric cultures were negative for acid-fast organisms. Other organisms and fungi were not demonstrated. Urine was negative. Bronchoscopic examination* in February, 1948, was reported negative.

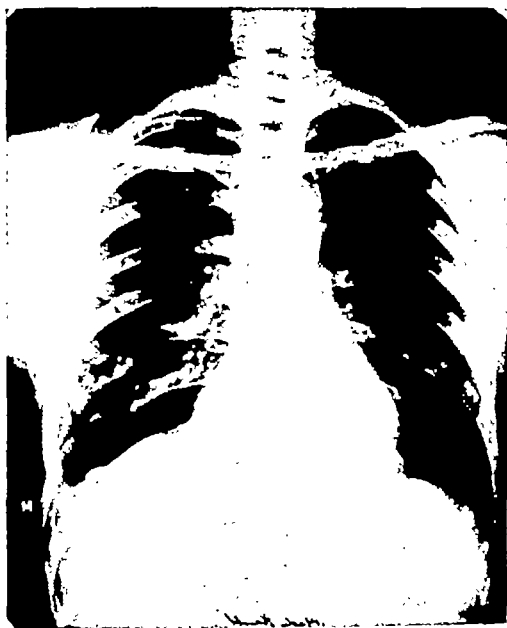


Fig. 8.—Follow-up roentgenogram (Feb. 1, 1949) taken ten months after operation shows no evidence of recurrence.

The patient was admitted to Grasslands Hospital on March 16, 1948, for surgical exploration. It was thought that the lesion seen roentgenographically was probably a benign tumor. Surgical exploration was done (by G. C. A.) on March 23, 1948, and a firm, well-circumscribed mass was found in the right middle lobe, extending through the interlobar fissure and involving a small portion of the right upper lobe. The hilum appeared free of involvement. A frozen section was made and a benign lesion was reported.† Segmental resection was done with removal of most of the right middle and a portion of the right upper lobe. The postoperative course was uneventful and the patient was discharged on April 2, 1948, the twelfth postoperative day. He is back at work, and there are no symptoms.

Pathology: The tumor mass measured 4 by 3 by 3 cm. and was firm. On section the surface was yellow gray in color and streaked with black pigmented strands. It was well demarcated from the surrounding lung tissue, but was not encapsulated. Microscopically the mass was composed of a dense framework of collagenous connective tissue in which was dispersed a variety of cellular forms. Plasma cells were numerous and in areas formed long cords or sheets. Russell fuchsin bodies, intracellular and extracellular, were frequently en-

*By Dr. M. T. Smith of New Rochelle, N. Y.

†By Dr. S. Gross, acting pathologist at Grasslands Hospital.

third of the esophagus with angulation to the left. At operation a tumor was found to involve the entire wall of the lower third of the esophagus, down to the cardia of the stomach. The lower esophagus and the cardia of the stomach were resected. The patient died on the fourth postoperative day of bilateral bronchopneumonia.

The same authors reported another patient, a woman, aged 56 years, who complained of pain beneath the xiphoid but no dysphagia. A large leiomyoma was found to involve the lower part of the esophagus with the greater part of the tumor in the posterior mediastinum and extending through the esophageal hiatus into the abdomen. The mucous membrane was intact. The tumor was excised locally except for a small portion attached to the pericardium which was left. Her postoperative course was uneventful. This tumor may have arisen from the stomach.

Harper and Tiscenko⁷ reported a male patient, aged 41 years, with a leiomyoma of the esophagus who for three years had had a feeling of tightness in the epigastrium following meals, which was relieved by vomiting. A submucosal tumor 6 by 3 cm. in size was found in the right posterior lateral wall of the middle third of the esophagus. The mucous membrane was intact and the pedicle was composed of avascular fibrous tissue. The tumor, a leiomyoma, was removed. The patient died on the fourth postoperative day and at post-mortem examination "wet lungs" were found.

Calmenson and Clagett⁸ reported a male patient, aged 52 years, who complained of substernal distress associated with slight difficulty in swallowing which occurred twice a year for ten years. Two leiomyomas were found in the wall of the esophagus at operation. They were located near the arch of the aorta and measured 2 and 6 cm. in diameter. Both tumors were removed without opening the esophageal mucosa. The patient's postoperative course was uneventful.

Schafer and Kittle⁹ reported a male patient, aged 46 years, who complained of epigastric distress and bilateral costovertebral angle pain with associated nausea and vomiting. There was no dysphagia. Roentgen examination of his thorax revealed a large rounded area of increased density in the lower posterior mediastinum displacing the esophagus to the right. At operation a lobulated, sharply delineated mass 7 by 7 by 10 cm. in size was found 6 cm. above the diaphragm. Removal of the tumor required excision of an ovoid section of the wall of the esophagus. The postoperative course was uneventful. Microscopically the tumor was a leiomyoma.

Hurwitz¹⁰ reported a male patient, aged 31 years, who entered the hospital for repair of an inguinal hernia. Routine roentgenogram of the chest revealed an ovoid shadow, 3 by 5 cm. in size just to the right of the midline and superimposed on the right heart border. Exploration of the chest was performed and a leiomyoma was found, one inch below the azygos vein protruding through the longitudinal muscle fiber of the esophagus and encircling three-quarters of it like a collar. The tumor was removed without opening the mucosa. The postoperative course was uneventful.

LEIOMYOMA OF THE ESOPHAGUS

ROLLIN A. DANIEL, JR., M.D., AND R. B. WILLIAMS, JR., M.D.

NASHVILLE, TENN.

BENIGN neoplasms of the esophagus are observed very infrequently. The leiomyoma is probably the most common benign tumor which arises in the esophagus. Patterson¹ reviewed the literature between the years 1717 and 1930 and found only 62 benign tumors reported, all of which were found at autopsy. The type of each tumor was not listed. Rose² reported 49 cases of leiomyoma of the esophagus found in the literature between 1797 and 1932 and added 1 of his own. All were found at autopsy. Moersch and Harrington³ report 44 cases of benign tumor of the esophagus found in 7,459 necropsies at Mayo Clinic. Thirty-two of these are leiomyomas. In 4,000 autopsies at the Vanderbilt University Hospital no case of leiomyoma of the esophagus was found.

It is evident that most leiomyomas of the esophagus do not produce symptoms. Moersch and Harrington³ report that in 11,000 patients complaining of dysphagia studied at Mayo Clinic only 15 benign tumors of the esophagus were found. Of these 2 were definite leiomyomas. In 3 others the diagnosis was listed as undetermined but probably leiomyoma. In none of the 32 leiomyomas found at autopsy at the Mayo Clinic were symptoms produced by the tumors.

Few cases of myoma of the esophagus have been discovered in the living patient. A search of the literature reveals that only 8 patients have been operated upon for removal of such a tumor. In 2 of these the tumor involved the cardia of the stomach as well as the esophagus, so their primary site could not be definitely determined.

Ohsawa⁴ reported a 43-year-old woman who complained of dysphagia of twenty-four hours duration and was found to have esophageal dilatation. In performing an esophagocardioplasty for relief of cardiospasm, an unsuspected submucosal myoma 1 cm. in diameter was found and enucleated. The patient survived the procedure.

Brock⁵ reported a male patient, aged 47 years, with leiomyoma of the esophagus, who complained of weakness, vomiting, pain in the right hypochondrium, loss of appetite, indigestion, and dragging sensation in the left side. At operation a lobulated mass measuring 10 by 7 cm. was found high on the lesser curvature of the stomach extending above and below the diaphragm. The distal end of the esophagus and the cardia of the stomach including the tumor was removed and an esophagogastrostomy performed. The patient recovered. This tumor involved the wall of both the esophagus and the stomach.

Harrington and Moersch⁶ reported a male patient, aged 38 years, with a leiomyoma of the esophagus who complained of nervousness and fatigue of four months' duration but no dysphagia. There was marked deformity of the lower

From the Department of Surgery, Vanderbilt University School of Medicine.
Received for publication June 13, 1949.

constant irritability in the esophagus about three inches above the diaphragm. The mucosal pattern appeared normal and there was no delay in passage of barium. Chest roentgenogram made on the eighth postoperative day showed a small amount of fluid at the base of the right lung and a slight residual collapse of the lower and middle lobes of the right lung.

The patient was discharged on the ninth postoperative day. Chest roentgenogram (Fig. 2) made about seven weeks following the operation revealed no abnormality other than a small amount of fluid at the base of the right lung and incomplete expansion of the lower and middle lobes of the right lung. She was and has remained asymptomatic.

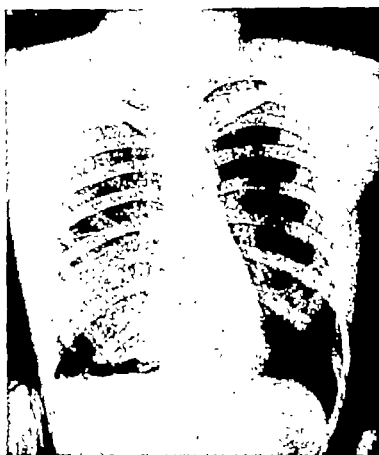


Fig. 2.—Roentgenogram made Feb. 16, 1948, seven weeks following operation.

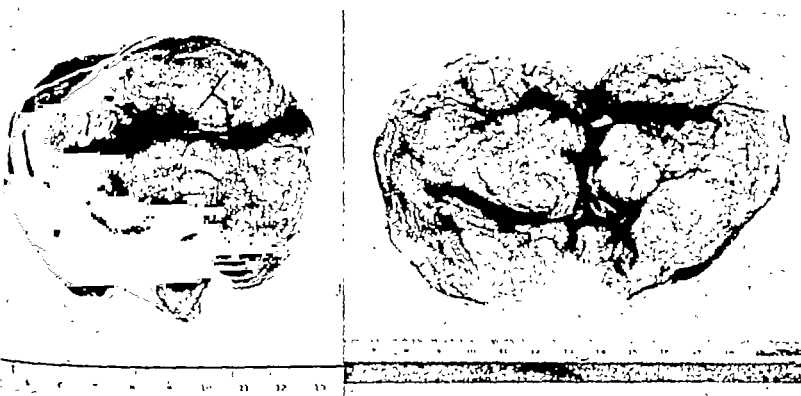


Fig. 3.—Photograph of the tumor following its removal.

CASE REPORT

A 38-year-old, white, married, female schoolteacher was admitted to the Vanderbilt University Hospital in December, 1947. She was told following routine chest examination six months previously that she had a growth in the right chest. She had had no cough, dysphagia, dyspnea, chest pain, nausea, vomiting, or any other symptoms referable to the chest. Physical examination revealed a well-developed and well-nourished woman. There were no abnormal physical findings. On chest roentgenogram (Fig. 1) a well-demarcated, homogeneous area of increased density measuring 6.5 by 6 cm. was seen in the right hemithorax overlying the medial aspect of the dome of the right diaphragm. Roentgen examination of the esophagus with barium showed some displacement of the esophagus to the left by the mass but no obstruction. There was a small paraesophageal hiatal hernia.

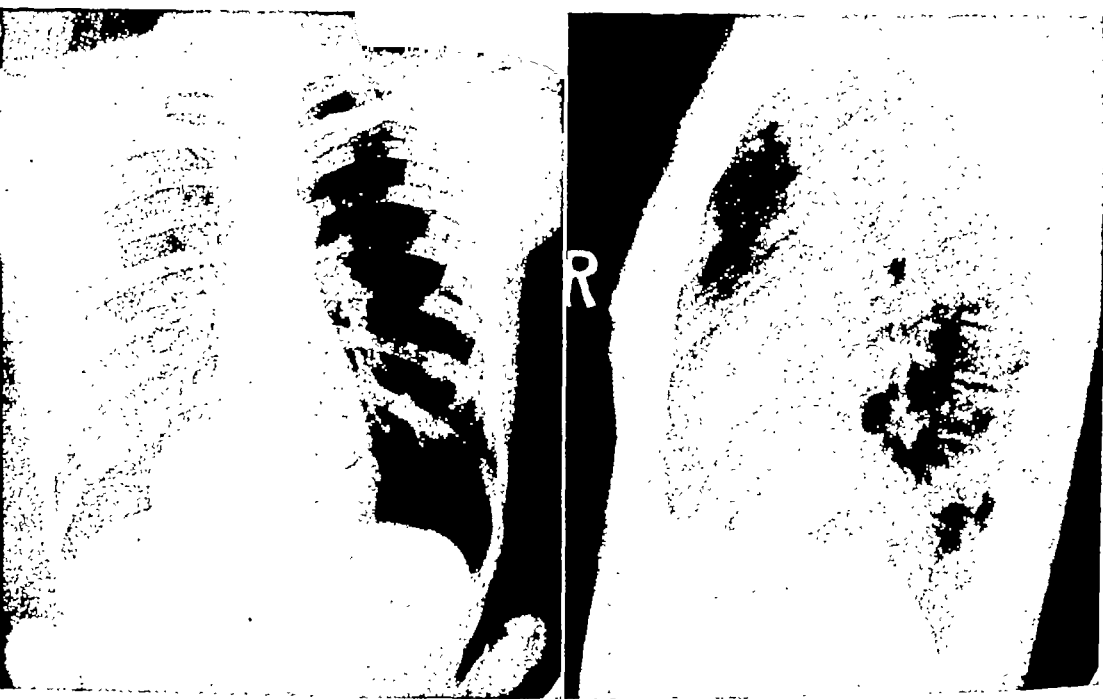


Fig. 1.—Roentgenograms made Dec. 29, 1947, one day prior to operation.

At operation on Dec. 30, 1947, a posterolateral incision was made over the ninth rib. A long segment of the ninth rib was removed subperiostally and the pleura opened. The lower lobe of the right lung was retracted upward and a firm, encapsulated, nodular mass about 7 cm. in diameter was present beneath the mediastinal pleura and posterior to the inferior pulmonary ligament. The mediastinal pleura was opened over the mass and the mass separated from the surrounding loose connective tissue. It was delivered into the pleural cavity and found to be attached to the esophagus by a pedicle 3 cm. long. It was easily freed from the wall of the esophagus by dividing the esophageal muscle and exposing the mucous membrane. The esophageal mucosa was not opened. Following removal of the tumor, the edges of the esophageal muscle were brought together with interrupted fine silk sutures. The lung was inflated and the wound of the chest wall closed in layers with interrupted fine silk sutures. The patient stood the procedure very well.

The postoperative course was uneventful. Wangensteen suction was instituted but discontinued on the second postoperative day and the patient started on a liquid diet. On the fifth day the patient was started on a soft diet which she took well. On the seventh postoperative day roentgen examination of the esophagus with barium revealed some in-

REFERENCES

1. Patterson, Ellen J.: *Benign Neoplasms of the Esophagus*, Pennsylvania M. J. 36: 244-246, 1933.
2. Rose, J. Dudfield: *Myomata of Esophagus*, Brit. J. Surg. 24: 297-308, 1936.
3. Moersch, Herman J., and Harrington, Stuart W.: *Benign Tumor of the Esophagus*, Ann. Otol., Rhin. & Laryng. 53: 800-817, 1944.
4. Ohsawa, T.: *The Surgery of the Esophagus*, Arch. f. jap. Chir. 10: 605-700, 1933.
5. Brock, R. C.: *Cardio-Esophageal Resection for Tumor of the Cardia*, Brit. J. Surg. 30: 146-150, 1942.
6. Harrington, S. W., and Moersch, H. J.: *Surgical Treatment and Clinical Manifestations of Benign Tumors of the Esophagus With Report of Seven Cases*, J. THORACIC SURG. 13: 394-414, 1944.
7. Harper, R. A. Kemp, and Tisceno, Eugene: *Benign Tumor of Esophagus and Differential Diagnosis*, Brit. J. Radiol. 18: 99-107, 1945.
8. Calmenson, Marvin, and Clagett, O. Theron: *Surgical Removal of Leiomyomas of the Esophagus*, Ann. J. Surg. 72: 745-747, 1946.
9. Schafer, Paul W., and Kittle, C. Frederick: *Esophageal Leiomyoma*, J. A. M. A. 133: 1202-1205, 1947.
10. Hurwitz, Alfred: *Leiomyoma of the Esophagus*, Surgery 25: 304-306, 1949.
11. Ewing, James: *Neoplastic Diseases*, ed. 4, Philadelphia, 1940, W. B. Saunders Co., p. 237.
12. Barazzuol, E.: *Contributo alla Studio dei Tumori Muscolari dell'esofago*, Arch. ital. d. mal. d. app. diger. 9: 41-50, 1940.

Examination of the gross specimen (Fig. 3) revealed a smooth, well-encapsulated, firm tumor with a lobulated surface and a pale, yellowish white color. The capsule was unbroken. On cut surface the tumor was yellowish in color and homogenous in appearance with grossly visible whorls. Some areas were firmer than others. No cysts were present.

Microscopic section (Fig. 4) of the tumor revealed strands of smooth muscle with long spindle-shaped cells with eosinophilic cytoplasm. The nuclei were short, stubby and uniform. No mitotic figures were present. This muscle was arranged in whorls. Masson stain revealed that connective tissue comprised grossly one-third of the total tissue.

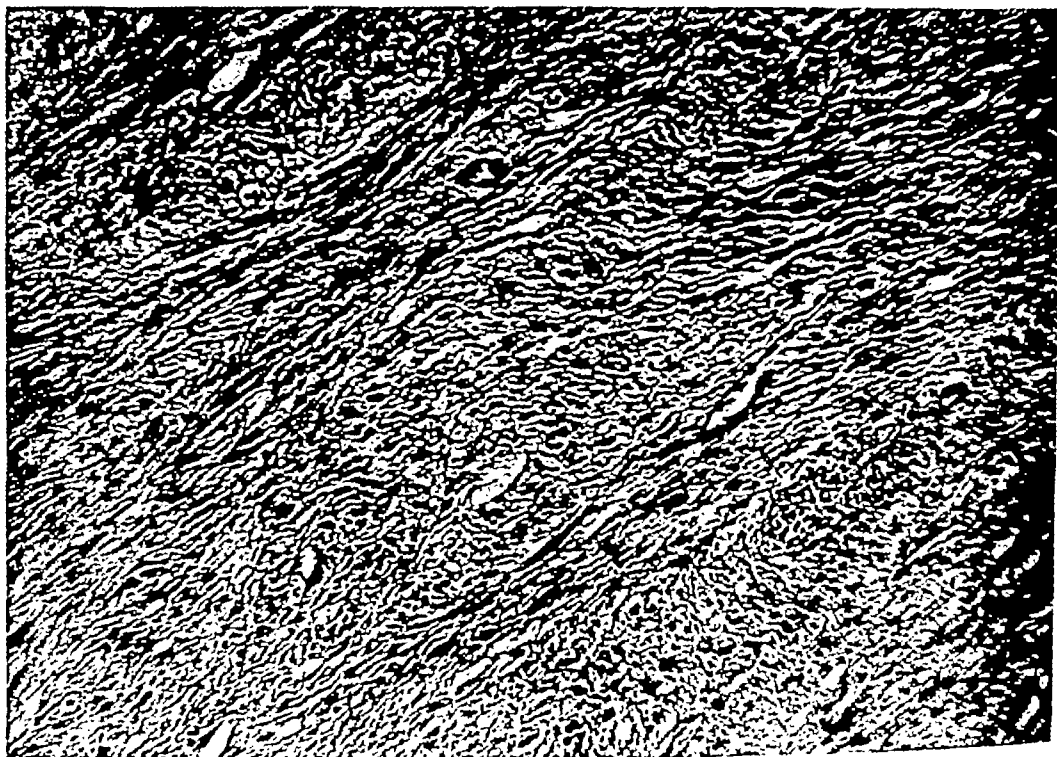


Fig. 4.—Photomicrograph of the tumor (about $\times 400$).

COMMENT

Ninety-two cases of leiomyoma of the esophagus have been reported in the literature.²⁻¹² One was complicated by islets of adenocarcinoma. Of the 92 patients, 8 were operated upon and 6 survived. In 2 of the 8, the tumor involved the cardia of the stomach as well as the esophagus so the primary site is not certain. Of the 6 patients in whom the diagnosis is certain, 4 survived operation.

Many of these tumors can be removed without opening the esophageal mucous membrane. In such cases the mortality rate should be extremely low. The tumors may arise at any level of the esophagus and they may be solitary or multiple but seldom are the cause of death. Rose,² however, reported a patient, aged 74 years, in whom multiple leiomyomas were the primary cause of death. These tumors have occurred in individuals of all ages but are most common in elderly people. They are more frequent in men.

The patient was admitted to Vanderbilt University Hospital and roentgen examination of the esophagus and stomach with barium was done. No filling defect or dilatation was present in either the esophagus or cardia of the stomach. Bronchoscopy revealed narrowing of the left bronchus as it passed into the lower lobe, but no ulceration or other abnormalities were seen.

On Nov. 15, 1947, under nitrous oxide, oxygen and ether inhalation anesthesia, exploration of the chest was made. A posterolateral incision was made over the left sixth rib and a long segment of this rib removed subperiosteally. The incision was extended through



Fig. 1.—Roentgenogram made Feb. 15, 1947, nine months prior to operation.



Fig. 2.—Roentgenograms made Nov. 8, 1947, one week prior to operation.

LEIOMYOMA OF THE LUNG

R. B. WILLIAMS, JR., M.D., AND ROLLIN A. DANIEL, JR., M.D.
NASHVILLE, TENN.

PRIMARY leiomyoma of the lung is rare. Review of the literature reveals that only four have been reported. Brahdy¹ reported a case in a young woman, aged 18 years, who was being studied as a tuberculous suspect. Roentgenogram of the chest showed a 1.6 cm., round area of density at the right lung base. X-ray examination of the chest made six months previously had shown no abnormality. The sputum became positive for acid-fast bacilli and in nineteen months a typical tuberculous infiltration was found in the right upper lung field. The mass at the right base gradually increased until three years later its diameter was approximately twice the original. Surgical exploration of the chest was done at this time and a "localized," round mass 2.5 cm. in diameter was found in the lower lobe of the right lung. The lower two-thirds of the lobe including the tumor was removed. The patient made an uneventful recovery. On cut surface the tumor was firm and white. Microscopic section revealed it to be a fibroleiomyoma.

Forkel² reported a case of leiomyoma of the lung found at autopsy on a woman, aged 63 years, who died of pneumonia. The "lemon-sized" mass was found in the apex of the upper lobe between the second and fourth ribs. Deussing³ reported a woman, aged 57 years, with multiple primary myomas of the lung found at autopsy. Franco⁴ reported an autopsy on a 56-year-old woman in whom a 13 by 11 by 9 cm. mass was found in the upper lobe of the left lung. This tumor was thought to be a leiomyoma.

CASE REPORT

On Jan. 18, 1947, an 8-year-old white girl was seen in the Vanderbilt University Hospital Pediatric Clinic complaining of a chronic cough of four years' duration. The cough was productive of clear mucoid sputum. There had been no pain, dyspnea, orthopnea, hemoptysis, weight loss, cyanosis, or retardation of growth. The child had done well in school and carried on normal physical activity without noticeable difficulty. Physical examination revealed some cervical lymph node enlargement and mild rhinitis. Physical examination of the thorax and lungs revealed no abnormal findings. Chest roentgenogram (Fig. 1) was made and interpreted as showing no abnormality. Allergy skin tests were made and several positive reactions obtained. Desensitization consisting of weekly injections of allergen was begun.

The cough after ten months was not improved. She was found at this time to have absence of breath sounds over the lower left thorax posteriorly. Roentgenogram of the chest was repeated and revealed a dense opacity measuring 10 by 7 cm. lying posteriorly over the mid-portion of the left diaphragm (Fig. 2). The original x-ray film of the chest was reviewed and the mass could be seen in the same location but was smaller and partially obscured by the heart shadow.

From the Department of Surgery, Vanderbilt University School of Medicine.
Received for publication June 13, 1949.

bronchus for almost its entire length was surrounded by the tumor and was obliterated by it. The cut surface of the tumor was pale yellow at the periphery with a central core which was light yellow and opaque with some diffuse calcification.

Microscopic sections (Fig. 5) showed the tumor to be composed of large whorls of spindle-shaped cells with elongated nuclei separated by collagenous connective tissue septa.

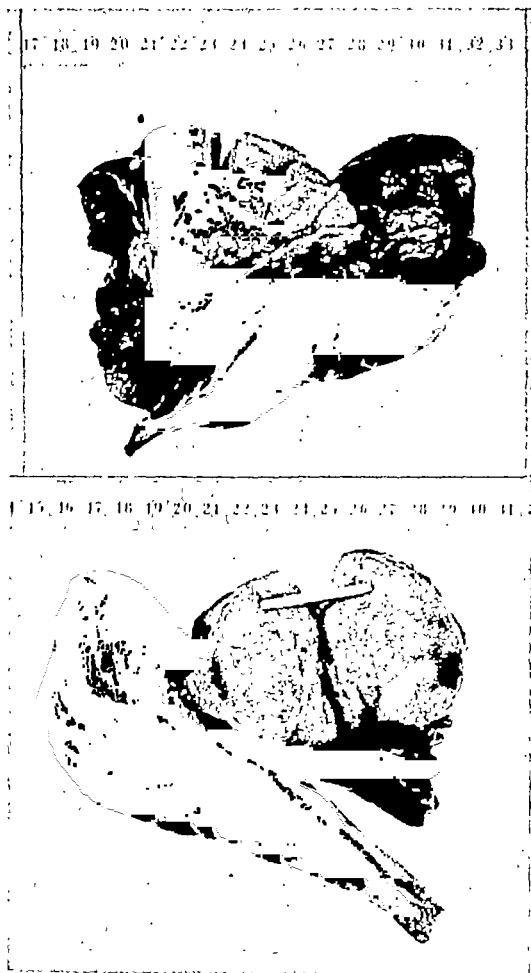


Fig. 4.—Left lung with leiomyoma in the lower lobe.

the periosteum and parietal pleura opening the hemithorax. A very large and extremely hard nodular tumor was found in the lower lobe of the left lung. This mass occupied at least one-half of the lower lobe, extending from the hilum laterally to the visceral pleura and downward to the diaphragm. The left lower lobe was densely adherent to the diaphragm over an area about 3 cm. in diameter and there were a large number of short dense adhesions between the lower lobe and the posterior chest wall. These adhesions were freed by sharp dissection and the lobe was freed from the diaphragm in the same manner. The fissure between the upper and lower lobes was quite distinct. The tumor was found to envelope the lower lobe bronchus and was firmly and densely adherent to it. What was thought to be the lower lobe bronchus was freed and occluded, but on inflating the lung this was found to be the upper lobe bronchus and it became evident that the lower lobe bronchus could not be exposed. Because of this and because the gross appearance of the tumor suggested that it might be malignant, pneumonectomy was performed. Following irrigation of the pleural cavity with saline, the wound of the chest wall was closed in layers with interrupted fine silk sutures.

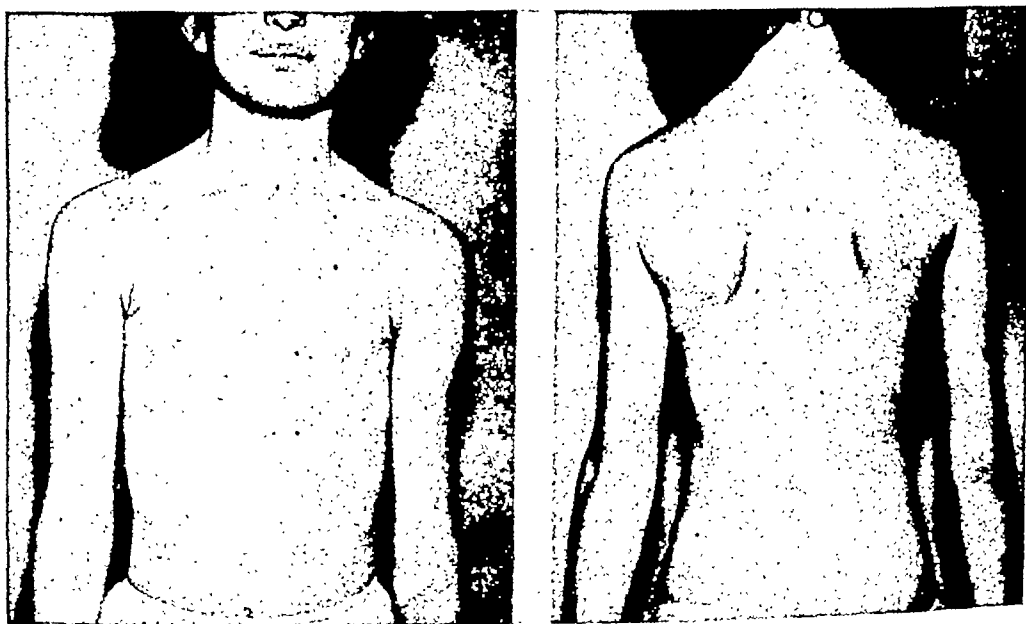


Fig. 3.—Photograph made April 16, 1949, seventeen months following pneumonectomy.

The postoperative course was uncomplicated. She reacted well and only on one occasion had rectal temperature over 100.4° F. On the second postoperative day thoracentesis was performed and 120 c.c. of bloody fluid and 80 c.c. of air were removed. She was afebrile after the third postoperative day. She received parenteral penicillin for six days postoperatively and was discharged from the hospital seven days after operation.

Since discharge the patient has had no serious difficulty. She has had occasional upper respiratory infections and her activities at school and at home have not been restricted. She has very little deformity of her thorax (Fig. 3). Roentgenogram of the chest made on Jan. 27, 1949, revealed the right lung to be clear. The lower one-half of the left hemithorax was opaque. The trachea and the heart were displaced markedly to the left.

Examination of the specimen (Fig. 4) after operation revealed the left lung to weigh 252 Gm. In the lower lobe was an oval, slightly nodular, firm, rubbery, pale white tumor measuring 7 cm. in diameter, presenting at the posterior, lateral, and inferior surfaces of the lung. The pleura covering the tumor was smooth and glistening. The lower lobe

AN APPARENTLY CONGENITAL BRONCHOESOPHAGEAL FISTULA PERSISTENT TO ADULT LIFE

DOUGLAS R. MORTON, M.D., JOHNSTON F. OSBORNE, M.D., AND
KARL P. KLASSEN, M.D.
COLUMBUS, OHIO

DURING the past decade significant progress in the field of thoracic surgery, together with the addition of chemotherapy and antibiotics to our therapeutic armamentarium, has accomplished the successful management of many cases of abnormal esophageal communications. These esophageal fistulas are classified into two main types: congenital and acquired. The latter is encountered most frequently, and may exist for a length of time without producing symptoms; in some instances it is even compatible with a relatively comfortable existence for a number of months. The congenital type invariably manifests itself with the first effort at swallowing, and if not recognized it is supposed uniformly to result in early death of the infant.

Monserrat¹ reported a series of 670 cases of esophageal fistula which illustrated the preponderance of the acquired type. Three hundred sixty-seven were associated with neoplasm, 222 were congenital, 41 were due to infection, and 40 were traumatic in their origin. Moersch and Tinney² in 1944, and Abbott³ in 1945, added additional cases; however, these reports were limited to the acquired type and revealed etiological factors similar to those of Monserrat. The most common causes of the acquired type of esophageal fistulas are esophageal and bronchial carcinoma, neoplasm of neighboring structures, for example, Hodgkin's disease, tuberculosis (empyema and peradenitis), syphilis, and trauma. Rarer cases have been reported secondary to esophageal stricture, diverticulum, esophagitis, actinomycesis, syphilitic aneurysm, vomiting, alcoholism, and cardiospasm.

Vogt⁴ classified congenital esophageal anomalies into three main types as follows:

- Type I. Complete absence of the esophagus.
- Type II. Atresia of the esophagus with an upper and lower esophageal segment, each ending in a blind pouch.
- Type III. Atresia of the esophagus with tracheoesophageal fistula.
 - (a) With fistula between upper segment and trachea.
 - (b) With fistula between lower segment and trachea.
 - (c) With fistula between both segments and trachea.

The congenital anomalies have been presented by Haight and Towsley,⁵ Ladd,⁶ and Holt, Haight, and Hodge.⁷ The latter group found 91.3 per cent of

From the Department of Research Surgery, The Ohio State University.
Aided by a Grant from the Mary Christie Curtis Fund for Surgical Research of The Ohio State University.
Received for publication June 24, 1949.

Some pallsading of cells and calcification was present. Where the spindle-shaped cells were cut at right angles, they, as well as their nuclei, appeared round. A large number of spaces lined with a single layer of endothelium and containing red blood cells were present throughout the tumor. With Masson's stain the whorls of spindle-shaped cells were brick red in color while the fibrous tissue septa were green. The tumor was surrounded by a thin fibrous tissue capsule.

The final diagnosis was leiomyoma of the lung.

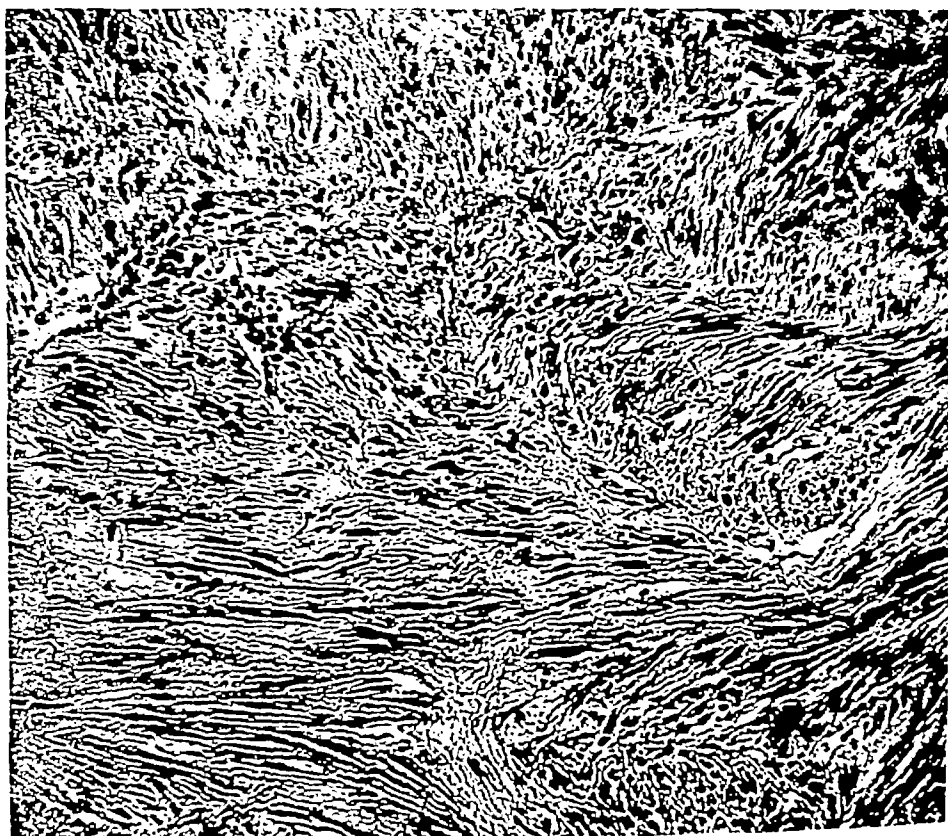


Fig. 5.—Photomicrograph of the tumor (about $\times 400$).

SUMMARY

A report of a patient with a leiomyoma of the lung treated by pneumonectomy is given. Four similar patients have been reported. One of these was successfully operated upon and in the other three, the tumors were found at autopsy.

REFERENCES

1. Brahdy, Leopold: Leiomyoma of Lung, *Am. Rev. Tuberc.* 43: 429-434, 1941.
2. Forkel, W.: Ein Fall von Fibromyom der Lunge, *Ztschr. f. Krebsforsch.* 8: 390-393, 1910.
3. Deussing, R.: Multiple Primary Myomas of Lung, Thesis, University of Munich, 1912.
4. Franco, Enrico Emilio: Sopra un rarissimo voluminoso leiomioma del polmone, *Tumori* 15: 27-42, 1929.

The patient was placed on systemic and aerosol penicillin two days preoperatively, resulting in a definite decrease in sputum production.

On the fifth hospital day under general endotracheal anesthesia a right posterolateral thoracotomy incision was made. After subperiosteal transection of the sixth and seventh ribs at their posterior angles, the chest was entered through the sixth intercostal space. On exploration there was moderate fibrosis and evidence of bronchiectasis in the posterior portion of the right lower lobe. Fine adhesions between the parietal and visceral pleura were divided. The pulmonary ligament was divided and the inferior pulmonary vein was ligated. During this process there was discovered a tubal structure about 5 cm. long and 5 mm. in diameter, which extended from the right anterolateral aspect of the esophagus at about the level of the ninth thoracic vertebra to the substance of the hilus of the right lower lobe posteriorly, about 1 cm. below the bifurcation of the right middle and lower lobe bronchi (Fig. 2). The tubal structure was isolated easily, doubly ligated with fine silk, and upon sectioning exhibited the characteristics of the gastrointestinal tract. The proximal stump was further closed with two over-end fine silk sutures at its origin in the esophagus. The right lower lobe was then removed, using the individual ligation technique. After instillation

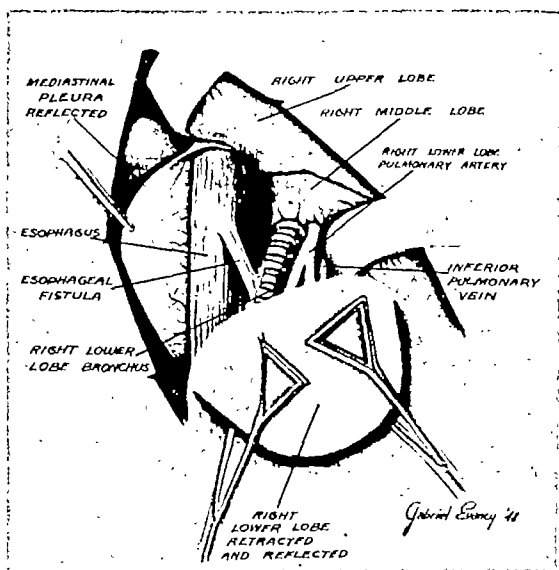


Fig. 2.—Diagrammatic illustration of relationship of structures as seen at surgery.

of 100,000 units of penicillin, the thorax was closed in a routine manner with a Pezzar catheter placed through a stab wound in the anterior axillary line. The drainage catheter was connected to a water-trap drainage bottle and removed forty-eight hours postoperatively, at which time the right lung was well expanded.

Study of the surgical specimen revealed a right lower lobe weighing 240 Gm. and measuring 10 by 10 by 8 cm. On its mediastinal surface there was a fistulous tract 2 cm.

a series of 46 cases to be of Type III (b). However, one of their cases presented particular diagnostic difficulty, for which they suggested the addition of another type to Vogt's classification: single tracheoesophageal fistula without atresia.

Because of the rarity of this condition, the following case of an apparent congenital bronchoesophageal fistula persistent to adult life and associated with unilobar bronchiectasis is presented.

CASE REPORT

J. G. S. (No. 484971), a 26-year-old white male university student was admitted to University Hospital June 16, 1948, complaining of cough of several years' duration. Following pertussis at the age of 4 years, he had noted a mild, persistent cough. Although he was seriously ill with pneumonia at 5 years of age, there was no definite increase in his cough. Other than his main complaint he remained in relatively good health until shortly after his entrance into the Army in 1942. After eight months' hospitalization for continuous colds, productive cough (1 cup per day), fever, chills, and weight loss of fifty pounds, he received a medical discharge from the Army, the diagnosis being bronchiectasis. Although there was some subsidence of complaints following discharge from the Army, he noted seasonal exacerbation of symptoms.



Fig. 1.—Admission bronchogram which reveals severe saccular bronchiectasis of the posterior basal segment of the right lower lobe. Arrow points to bronchoesophageal fistula.

On physical examination the essential findings were limited to the chest, there being slight dullness and decreased breath sounds in the right posterior base. All laboratory data were within normal limits. Chest roentgenograms revealed an increased density in the region of the right posterior basal segment. There was a shift of the heart to the right with slight elevation of the diaphragm. A bronchogram showed severe saccular bronchiectasis of the posterior basal segment of the right lower lobe. The changes in the anterior basal segment were less severe (Fig. 1).

in length and 5 mm. in diameter arising from the right lower lobe bronchus. The fistula was opened and was seen to contain a collection of mucus and an undigested pebble rind. The remainder of the lung was atelectatic with dilatation of the main bronchi. Microscopic sections revealed the fistulous tract to be composed of smooth muscle tissue and lined with thin stratified squamous epithelium (Fig. 3). The remainder of the lobe revealed the microscopic appearance of advanced bronchiectasis.

A review of the history was undertaken with specific questioning of the patient and his relatives. The mother had hyporemesia gravidarum during carriage of the patient. There was no difficulty in nursing or swallowing as an infant. The patient volunteered the following symptomatology which was previously withheld because other physicians had labeled him as a psychoneurotic.

During his eight months' hospitalization in an Army hospital he complained of continuous upper respiratory infection, congested nose, general malaise, myalgia, and chills and fever. It was at this time that the cough and sputum production were definitely increased, and it was noted that the paroxysms of coughing and choking sensations were initiated by ingesting fluids. "It seemed as if something was dripping into the lungs which had to be coughed up." On many occasions he coughed up coffee grounds, pea hulls, and bread crumbs. There was no associated vomiting or regurgitation. These paroxysms were more frequent when lying down, especially when on the left side.

The patient was discharged greatly improved on the thirteenth hospital day. Six weeks postoperatively he was asymptomatic and a roentgenogram revealed minimal postoperative pleural thickening (Fig. 4). During the year since surgery the symptoms have completely subsided; he has gained weight, and is doing well in his schoolwork.

DISCUSSION

The preceding case presents two pathologic entities, a congenital bronchoesophageal fistula and right lower lobe bronchiectasis. We assume the fistula to be congenital inasmuch as the tract was such a well-defined tubal structure that when first seen in the hilar substance it was mistaken for an accessory blood vessel. However, transection of the tube exhibited the characteristics of the gastrointestinal tract and was followed proximally to its entrance into the esophagus. The adhesions encountered at surgery, although prominent over the bronchiectatic lobe, were absent over the proximal one-half of the fistulous tube.

Although cough had been present since infancy, the typical symptoms of congenital esophageal fistula did not become manifest until the patient had reached adult years. Unfortunately these typical symptoms were loosely labeled "psychoneurotic," and the patient hesitated before volunteering the information which aided in making a correct diagnosis of the condition as found at surgery.

SUMMARY

1. A case of congenital bronchoesophageal fistula with secondary bronchiectasis in an adult is presented.
2. The onset of the typical symptomatology of a congenital bronchoesophageal fistula did not become manifest until adult life. Right lower lobectomy and closure of the esophageal fistula resulted in complete recovery.

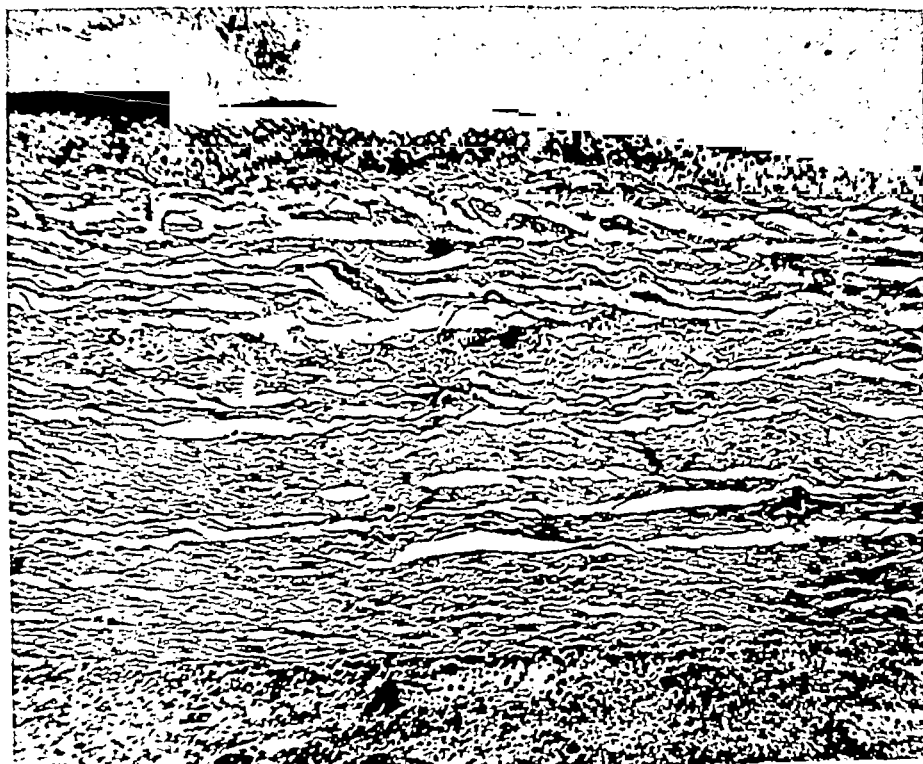


Fig. 3.—Microphotograph ($\times 10$) of bronchoesophageal fistula. Note squamous-cell epithelium.



Fig. 4.—Roentgenogram of chest six weeks following right lower lobectomy.

A NEW METHOD OF AUTOMATIC CONTROLLED RESPIRATION

MILTON H. ADELMAN, M.D., ROBERT A. BERMAN, M.D.,* AND
ARTHUR S. W. TOUROFF, M.D.
NEW YORK, N. Y.

CONTROLLED respiration may be defined as an anesthetic technique in which apnea is intentionally induced and respiration then maintained by intermittent inflation of the lungs. First described by Guedel and Treweek¹ in 1934, it now is considered by many surgeons and anesthesiologists to be a valuable aid in thoracic surgery.²⁻⁵ The development of mechanical devices to maintain controlled respiration automatically was inevitable and the Scandinavian workers have been pre-eminent in the field. Thus, in 1940, Crafoord⁶ of Stockholm described the Crafoord-Frenckner spiropulsator and its successful application to thoracic surgery. Moersch⁷ of Denmark, in 1947, reported a respirator of original design for automatic control of respiration. Mautz,⁸ one of the pioneers in our own country, presented his experiences with a mechanical respirator for automatic and augmented breathing in 1941.

In our approach to the problem of automatic controlled respiration, we hold the belief that there was need for an apparatus less complicated than those described by the aforementioned workers. Thus, when Harroun and co-workers⁹ reported their pentothal-curare apneic technique in transthoracic surgery, it occurred to us that some simple automatic device could be adapted to the Harroun technique in order to maintain controlled respiration more effectively than by manual methods. The pneumatic balance resuscitator appeared to be such a device.

The pneumatic balance resuscitator (PBR)[†] is a light, simple, sturdy apparatus which permits of effective cleansing and sterilization. It was developed by Burns⁹ and studied extensively by Motley and co-workers.¹⁰ In brief, the PBR consists of a differential pressure valve which converts continuous positive pressure into intermittent positive pressure. Changes in positive pressure are obtained through a pressure-regulating device attached to the source of continuous pressure (oxygen cylinder). The differential pressure valve requires no adjustment. A nebulizer can be incorporated into the PBR for humidification of the dry oxygen gas and for the nebulization of antibiotics during anesthesia.¹¹

We have used the pentothal-curare apneic technique in conjunction with the PBR in twenty cases. A brief description of this anesthetic technique and some clinical observations follow.

From the Department of Anesthesiology and the Surgical Service of Dr. Arthur S. W. Touroff, The Mount Sinai Hospital.

Received for publication May 23, 1949.

*Now at St. Joseph's Hospital, Far Rockaway, N. Y.

†The device is manufactured by the Mine Safety Appliance Company, Pittsburgh, Pa. It is marketed as the M.S.A. Pneophore and has received acceptance, with reservations, by the Council of Physical Medicine of the American Medical Association.

REFERENCES

1. Monserrat, J. L.: Fistulas tuberculosas esofago-traqueo-bien quicas, *Rev. Asoc. med. argent.* 55: 438, 1941.
2. Moersch, H. J., and Tinney, W. S.: Fistula Between the Esophagus and the Tracheo-bronchial Tree, *M. Clin. North America* 28: 1001, 1944.
3. Abbott, O. A.: Abnormal Esophageal Communications: Their Types, Diagnosis and Therapy, *J. THORACIC SURG.* 14: 382-392, 1945.
4. Vogt, E. C.: Congenital Esophageal Atresia, *Am. J. Roentgenol.* 22: 463, 1929.
5. Haight, C., and Towsley, H. A.: Congenital Atresia of the Esophagus With Tracheo-Esophageal Fistula, *Surg., Gynec. & Obst.* 76: 672-688, 1942.
6. Ladd, W. E.: The Surgical Treatment of Esophageal Atresia and Tracheo-Esophageal Fistula, *New England J. Med.* 230: 625-637, 1944.
7. Holt, J. F., Haight, C., and Hodges, F. J.: Congenital Atresia of the Esophagus and Tracheo-Esophageal Fistula, *Radiology* 47: 457-470, 1946.

The Journal of Thoracic Surgery

Vol. 19

JUNE, 1950

No. 6

Original Communications

BRONCHOSPIROMETRY IN THE LATERAL DECUBITUS POSITION

EMIL ROTHSTEIN, M.D.,* FRANCIS B. LANDIS, M.D.,† AND
BENJAMIN G. NARODICK, M.D.,‡ WOOD, WIS.

INTRODUCTION

SINCE the original work by Jacobaeus¹ and the development by Gebauer² and Zavod³ of double lumen soft rubber bronchial catheters with occlusive balloons, bronchspirometry has become an accepted procedure for analyzing the respiratory and ventilatory functions of the individual lungs. This report is a study of bronchspirometry in the lateral decubitus position as one of a series of studies in pulmonary and respiratory physiology. It records our observations on a number of patients in whom bronchspirometry was performed in one or both lateral decubitus positions immediately after a satisfactory tracing was obtained with the patient supine. We have studied eleven patients by this method. In six we have recordings in both lateral decubitus positions, and in five in the right lateral decubitus alone. Six of these patients had uncomplicated pulmonary tuberculosis (Table I). In one there was a unilateral phrenic paralysis; one had unilateral artificial pneumothorax plus contralateral phrenic paralysis; one had a nonobstructing carcinoma of the left lower lobe bronchus; one had a mediastinal cyst; and one had had a right lower lobectomy for pulmonary tuberculosis associated with right phrenic paralysis.

TECHNIQUE

The patient is prepared by receiving $1\frac{1}{2}$ gr. of pentobarbital sodium by mouth one hour before bronchspirometry. One-half hour before the procedure, a hypodermic injection of 100 mg. of demerol and $\frac{1}{32}$ gr. of atropine sulfate is administered. Anesthesia of the throat and larynx is secured by local application of 2 per cent pontocaine.

From the Tuberculosis and Surgical Services, Veterans Administration Hospital, Wood, Wis.

Sponsored by the Veterans Administration and published with the approval of the Chief Medical Director. The statements and conclusions published by the authors are a result of their own study and do not necessarily reflect the opinion or policy of the Veterans Administration.

Received for publication Aug. 22, 1949.

*Chief, Tuberculosis Service, Veterans Administration Center, Dayton, Ohio.

†Staff member, Tuberculosis Service, Veterans Administration Hospital.

‡Resident in Surgery, Department of Surgery, Veterans Administration Hospital.

cases. Operating conditions were excellent. It must be emphasized that the work is still in its early phases and that the technique described should be executed only by a skilled anesthesiologist.

SUMMARY

A new technique of automatic controlled respiration is described. The method utilizes pentothal-curare anesthesia and the pneumatic balance resuscitator. A brief clinical report is presented.

REFERENCES

1. Guedel, A. E., and Treweek, Q. N.: Ether Apnoeas, *Anesth. & Analg.* 13: 263-264, 1934.
2. Nosworthy, M. D.: Anesthesia in Chest Surgery With Special Reference to Controlled Respiration and Cyclopropane, *Proc. Roy. Soc. Med.* 34: 479-506, 1941.
3. Neff, W., Phillips, W., and Gunn, G.: Anesthesia for Pneumonectomy in Man, *Anesthesiology* 3: 314-322, 1942.
4. Orton, R. H.: Controlled Respiration, *M. J. Australia* 2: 255-259, 1947.
5. Crafoord, C.: Pulmonary Ventilation and Anesthesia in Major Chest Surgery, *J. THORACIC SURG.* 9: 237-253, 1940.
6. Mautz, F. R.: A Mechanism for Artificial Pulmonary Ventilation in the Operating Room, *J. THORACIC SURG.* 10: 544-550, 1941.
7. Moersch, E. T.: Controlled Respiration by Means of Special Automatic Machines as Used in Sweden and Denmark, *Proc. Roy. Soc. Med.* 40: 603-607, 1947.
8. Harroun, P., and Hathaway, H. R.: The Use of Curare in Anesthesia for Thoracic Surgery, *Surg., Gynec. & Obst.* 82: 229-231, 1946.
9. Burns, H. I.: Pneumatic Balance Resuscitator, *Air Surgeon's Bull.* 2: 306, 1945.
10. Motley, H. L., Cournand, A., Eckman, M., and Richards, D. W., Jr.: Physiological Studies on Man With Pneumatic Balance Resuscitator "Burns Model," *J. Aviation Med.* 17: 431, 1946.
11. Motley, H. L., Lang, L. P., and Gordon, B.: Use of Intermittent Positive Pressure Breathing Combined With Nebulization in Pulmonary Disease, *Am. J. Med.* 5: 853-856, 1948.

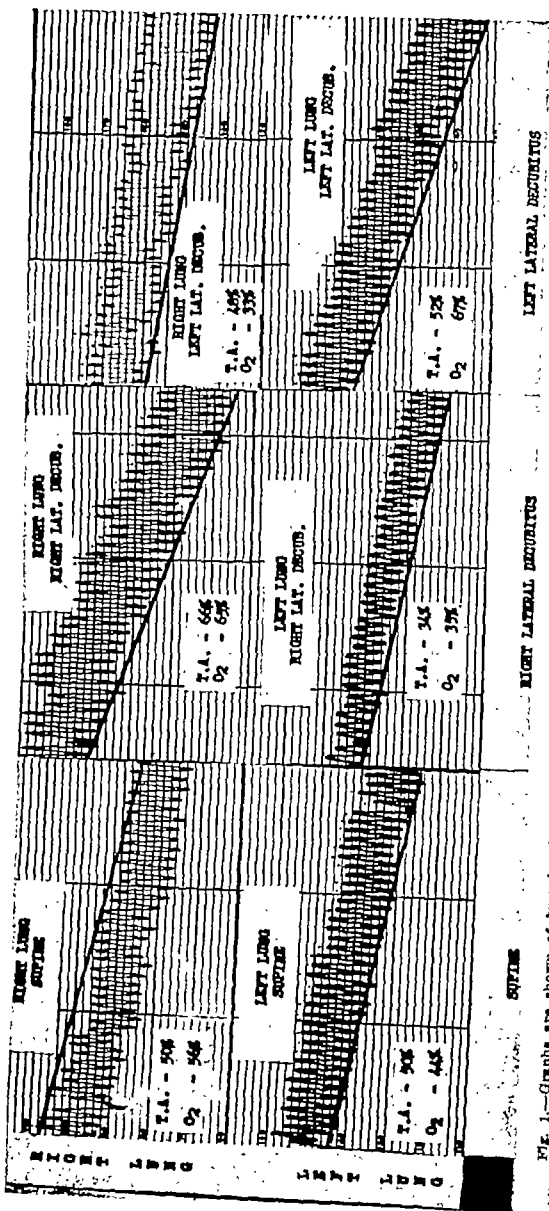


FIG. 1.—Graphs are shown of bronchospirometric readings made on one patient in the supine, the right lateral decubitus, and left lateral decubitus positions. With the patient in the supine position, the right lung performed 60 per cent of the total ventilation and 56 per cent of the total oxygen absorption. When the patient was placed on the right side, the tidal air increased to 66 per cent and the oxygen consumption to 66 per cent of the total. With the patient lying on the left side, the tidal air on the right dropped to 48 per cent of the total and the oxygen absorption dropped to 53 per cent of the total.

With the catheter in the trachea and its tip in the left main bronchus (verified by means of fluoroscopy) the double-barreled catheter was connected to two Benedict-Roth spirometers with the tanks containing oxygen. The balloons were then inflated and a graph was obtained from each lung by means of continuous, simultaneous tracings on two revolving drums while the patient was in a supine position. Without displacing the catheter or deflating the balloons, the patient was helped to turn on the right side. A pillow was placed under the head. In all other respects the conditions remained unchanged. Readings were again taken from each lung with the patient in this position for a period of from two to five minutes. The shorter interval was used only when the respirations were smooth and uniform in size with an even slope indicating a smooth oxygen absorption curve. The patient was then assisted to roll on the left side and further graphs were secured in this position.

TABLE I

CASE	DIAGNOSIS
1	Pulmonary tuberculosis, right phrenic paralysis, left pneumothorax
2	Pulmonary tuberculosis, minimal, left
3	Pulmonary tuberculosis, far advanced, left lung only
4	Pulmonary tuberculosis, minimal, right
5	Carcinoma, left lower lobe bronchus
6	Pulmonary tuberculosis, minimal with left phrenic paralysis
7	Mediastinal cyst, right
8	Pulmonary tuberculosis, moderately advanced, bilateral
9	Pulmonary tuberculosis, far advanced, predominantly right-sided
10	Pulmonary tuberculosis, post right lower lobectomy and right phrenic paralysis
11	Pulmonary tuberculosis, far advanced, predominantly right-sided

The total oxygen consumption (the amount used per minute by both lungs) and the differential oxygen consumption of the right and left lungs, the total tidal air and its division between the two lungs, the ventilation equivalents, and in some cases the vital capacities of each lung were compared with the patient supine and in the right and left lateral decubitus positions. In making these comparisons, the slope of the graph (see Fig. 1) was used to determine the oxygen absorption. In making tidal air determinations, consecutive, individual respirations were measured with calipers over a two- or three-minute interval. Identical respirations by the right and left lungs were thus measured and an average determined for each lung.

Not all patients who have bronchspirometric studies are suitable for these special determinations. As the procedure is prolonged there may be increasing cough and secretion, and in some the catheter becomes partly dislodged as the patient is turned on the side. The nervous temperament of the patient and his willingness to cooperate are important factors in securing satisfactory tracings.

RESULTS

Application of logic to our knowledge of pulmonary and respiratory anatomy and physiology would seem to dictate that the lung which is down should do less of the breathing. Our results, however, would indicate that exactly the opposite is true.

disease and most showed alterations from this normal range even while in the supine position. Table III illustrates these percentages for the patients when supine, and in each lateral decubitus position. It will be noticed that in every instance the under lung shows a definite increase in the amount and percentage of the total oxygen consumed and that in some cases this was quite marked. The percentage absorbed by the upper lung was diminished in every case; the amount was often decreased as well.

TABLE III. DIFFERENTIAL O₂ ABSORPTION IN CUBIC CENTIMETERS PER MINUTE AND PER CENT OF TOTAL

CASE	SUPINE				RIGHT LATERAL				LEFT LATERAL			
	VOLUME		PER CENT		VOLUME		PER CENT		VOLUME		PER CENT	
	RIGHT	LEFT	RIGHT	LEFT	RIGHT	LEFT	RIGHT	LEFT	RIGHT	LEFT	RIGHT	LEFT
1*	91	105	32	08	176	238	42	58				
2	104	94	52	48	321	197	02	38				
3	111	124	47	53	235	150	00	40				
4	145	117	56	44	238	180	57	43	138	210	40	60
5	210	124	03	37	280	119	70	30	131	161	48	55
6†	106	130	56	44	249	135	05	35	107	221	33	67
7	197	202	48	52	200	152	60	34	187	294	39	61
8	102	200	40	51	388	186	68	32				
9	93	137	41	59	104	90	08	32				
10*	98	232	30	70	138	235	37	63	62	290	18	82
11	104	161	39	61	150	149	50	50	62	194	25	75

*Right phrenic paralysis.

†Left phrenic paralysis.

Total Tidal Air.—The total tidal air with the patient in the supine position averaged 636 c.c. with a range from 545 to 894 c.c. Little change in this total (Table IV) was noted with the patient in the right lateral decubitus but there was a moderate increase when he lay upon the left side (right lateral decubitus 659 c.c., left lateral decubitus 696 c.c.). The respiratory rate and, therefore, the minute ventilation were essentially unchanged.

TABLE IV. TOTAL TIDAL AIR IN CUBIC CENTIMETERS PER MINUTE

CASE	SUPINE	RIGHT LATERAL	LEFT LATERAL
1	002	503	
2	023	083	
3	053	087	
4	052	049	022
5	581	081	710
6	606	050	042
7	707	003	757
8	609	098	
9	545	572	
10	894	014	883
11	407	408	500
AVERAGE	636	659	696

Differential Tidal Air (Table V).—In the normal individual the right side ventilates about 55 per cent of the total tidal air, and the left side about 45 per cent of the total. In the presence of pulmonary disease with the subject supine, the differential ventilation is usually much less affected than the oxygen consumption. An abnormal lung may contribute its normal percentage of the total ventilation. This can be seen by a comparison between the first four

Total Oxygen Consumption.—Absolute readings of oxygen consumption by both lungs are not of much value when bronchspirometry is performed because of the lack of basal conditions, the addition of sedation, and the extra effort which is needed to breathe through the bronchspirometric catheter. These conditions do not influence the proportion of the total oxygen breathed by each lung. Therefore, in most bronchspirometric studies only the relative percentage of oxygen absorbed by the individual lung is measured. However, for the purposes of this study, it was felt that the total oxygen consumption should be measured as well as the percentage absorbed by each lung so that a comparison could be made between this figure in each of the three positions (Table II). With the patient supine, the total oxygen consumption for both lungs per minute varied from 235 c.c. to 399 c.c. with an average of 303 c.c. When the subject lay upon the right side the total oxygen consumption for both lungs varied from 284 c.c. to 574 c.c., with an average of 409 c.c. The total oxygen consumption increased in every case in this position, ranging from 35 c.c. to 220 c.c., with an average increase of 106 c.c. or 34 per cent. One hundred and three cubic centimeters represented the average increase in the oxygen consumption of the right or under lung, and 3 c.c. represented the average increase in the oxygen consumption of the left lung, but there was considerable variation between individual cases. In several the oxygen consumption of the left lung showed an appreciable decrease. Six patients were studied in the left lateral decubitus position. These patients had an average oxygen consumption of 315 c.c. while supine and 343 c.c. while lying on the left side, an average increase of 9 per cent. This was due in each case to an increased oxygen absorption by the left or under lung associated with a drop in the oxygen absorption of the right lung. In all but one instance the total oxygen consumption while lying on the left side was less than that observed with the patient lying on the right side, and in two instances it was less than while he was lying supine.

TABLE II. TOTAL OXYGEN ABSORPTION IN CUBIC CENTIMETERS PER MINUTE

CASE	SUPINE	RIGHT LATERAL	LEFT LATERAL
1	286	414	
2	198	518	
3	235	394	
4	262	418	348
5	340	399	292
6	296	384	328
7	399	442	481
8	392	574	
9	231	284	
10	330	373	352
11	265	299	256
AVERAGE	303	409	343

Differential Oxygen Consumption.—This term is used to describe the proportions of oxygen used by the individual lungs. The usual findings in normal subjects with the patient supine are as follows: the right lung absorbs 55 per cent of the total oxygen consumption and the left 45 per cent with a range of 5 per cent in either direction. All of the present group studied had pulmonary

CONCLUSIONS

Bronchspirometry done in the lateral decubitus position and compared with readings obtained with the patient in the supine position reveals a slightly increased percentage of ventilation and a moderate to marked increase in percentage of oxygen consumption in the under lung.

ADDENDUM

As a result of this study, an additional question was raised. Is the oxygen consumption increased in the lateral decubitus position in patients not undergoing bronchspirometry?

Ten patients were studied in the following manner: Oxygen consumption was determined in the supine, right lateral decubitus, and left lateral decubitus positions using the Benedict-Roth metabolism apparatus. These patients rested at least fifteen minutes prior to the determination, but were not in a basal state.

In each instance an increase in oxygen consumption was noted in the lateral decubitus position as compared with the supine position. The average O_2 consumption in the supine position was 306 c.c. O_2 per minute. While lying on the right side, the O_2 consumption increased to 380 c.c. per minute, an increase of 26 per cent. While lying on the left side, the O_2 consumption increased to 374 c.c. per minute, an increase of 22 per cent.

These findings are similar to those noted in patients undergoing bronchspirometry in the lateral decubitus position. The exact explanation for the increased utilization of oxygen with the patient in the decubitus position is not immediately apparent. A further study of this problem is being undertaken at this institution, and the results of this study will be reported at a later date.

REFERENCES

1. Jacobaeus, H. C.: J. THORACIC SURG. 7: 235, 1938.
2. Gebauer, P. W.: J. THORACIC SURG. 8: 674, 1939.
3. Zavod, W. A.: J. THORACIC SURG. 10: 27, 1940.
4. Pinner, Max, Leiner, George C., and Zavod, W. A.: Ann. Int. Med. 22: 704, 1945.
5. Frenckner, P., and Björkman, S.: Proc. Roy. Soc. Med. 30: 477, 1936.

congested under lung presents a larger capillary bed for increased oxygen absorption. This increased efficiency is also reflected in the diminished ventilation equivalent.

Total Tidal Air.—This showed 4 per cent increase with the patient in the right lateral decubitus position and 10 per cent increase in the left lateral decubitus. These changes are probably within the range of experimental error.

Differential Tidal Air.—The differential tidal air in nearly every instance showed an increase in the percentage breathed by the under lung as compared with those proportions noted with the patient supine. It is difficult for us to understand why this should occur. In three of the cases there was paralysis of the diaphragm. Despite this fact the lung on the paralyzed side showed increased ventilation (as well as increased oxygen absorption) when the patient lay upon that side. The one patient who had artificial pneumothorax did not have readings made while lying on the pneumothorax side.

Ventilation Equivalent.—Changes in the ventilation equivalent are merely reflections of the changes in oxygen absorption and minute ventilation and need no further discussion.

As a clinical application of the findings described previously, we should like to note that on a number of occasions we have seen patients who were dyspneic due to unilateral pulmonary disease. In several instances such patients have preferred to lie on the uninvolved side and have found that the dyspnea vanished or was less distressing in this position. It is possible that this observation can be explained by the increased ventilation and increased oxygen absorption described here.

Vital Capacity.—The changes in the vital capacity were not constant.

SUMMARY

1. Bronchspirometry in the supine and right lateral decubitus positions has been performed in eleven patients. Six of these also had determinations made while lying in the left lateral decubitus position.

2. The total oxygen consumption increased an average of 34 per cent while lying on the right side and 9 per cent while lying upon the left. This was due almost entirely to an increase in the oxygen absorption by the under lung. In the majority a simultaneous decrease was noted in the oxygen absorption of the upper lung but in some this lung also showed increased oxygen absorption.

3. A definite increase in the percentage of oxygen consumption was noted in every instance in the under lung.

4. A slight increase in the total tidal air was noted in the lateral as compared with the supine position.

5. A slight but definite increase in the percentage of tidal air was noted in the under lung in the lateral position.

6. The ventilation equivalent, an index of respiratory efficiency, was decreased in the under lung, indicating increased efficiency.

7. The vital capacity showed inconstant changes.

small vacuoles. In the larger cells, which were fewer in number, the cytoplasm was almost completely occupied by a single large vacuole. The nuclei were small, deeply chromatic, and usually centrally located except in the larger, unilocular cells where they were eccentrically placed.

In other parts of the tumor, which grossly appeared myxomatous, the cell walls had undergone dissolution leaving only scattered fusiform nuclei on a pale, fibrillary, basophilic background. Small groups of lymphocytes and plasma cells were present throughout the entire neoplasm, more predominantly in the degenerated areas.

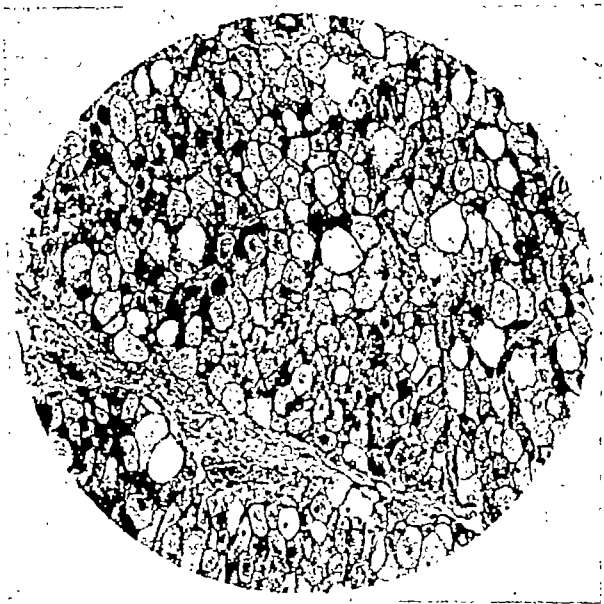


Fig. 5.—Photomicrograph illustrating the hibernoma cell type: ovoid and irregular, packed with fine and coarse cytoplasmic vacuoles, and possessing a small dark nucleus. Note the variations in vacuolar size and the resemblance of this tissue to immature fat.

Special stains revealed the cytoplasmic vacuoles to possess the tinctorial reactions of lipid material.

Diagnosis.—Atypical lipoma derived from the human homologue of the hibernating gland (hibernoma).*

Chemical Analysis.—For comparison analysis† was done on a large subcutaneous lipoma simultaneously with the hibernoma. Findings were as shown in Table I.

The following tests were determined on the triglyceride of these tumors and are reported on that basis:

	LIPOMA	HIBERNOMA
Iodine number	50.9	55.0
Acid value	3.1	5.3
Melting point	22° C.	1° C.

*Dr. Brines confirmed this diagnosis and stated it to be identical with the neoplasm in his patient.^{5, 6}

†This chemical analysis was done through the courtesy of Dr. E. L. Boley, Armour and Company, Chicago, Ill.

left lung. These were ligated and divided mobilizing the internal surface of the mass. To minimize the likelihood of recurrence the tumor was resected en bloc with the attached chest wall containing segments of the third, fourth, fifth, and sixth ribs.

The chest was closed by anatomic approximation of its layers. The thoracic cavity was not drained. The operative procedure required one hour and thirty-two minutes. Whole blood, 1,000 c.c., was administered intravenously.

The postoperative course was uneventful with the exception of 450 c.c. of a sero-sanguineous fluid which was removed by thoracentesis on the third postoperative day. The patient was discharged on the eleventh day following operation. She has been completely asymptomatic since.

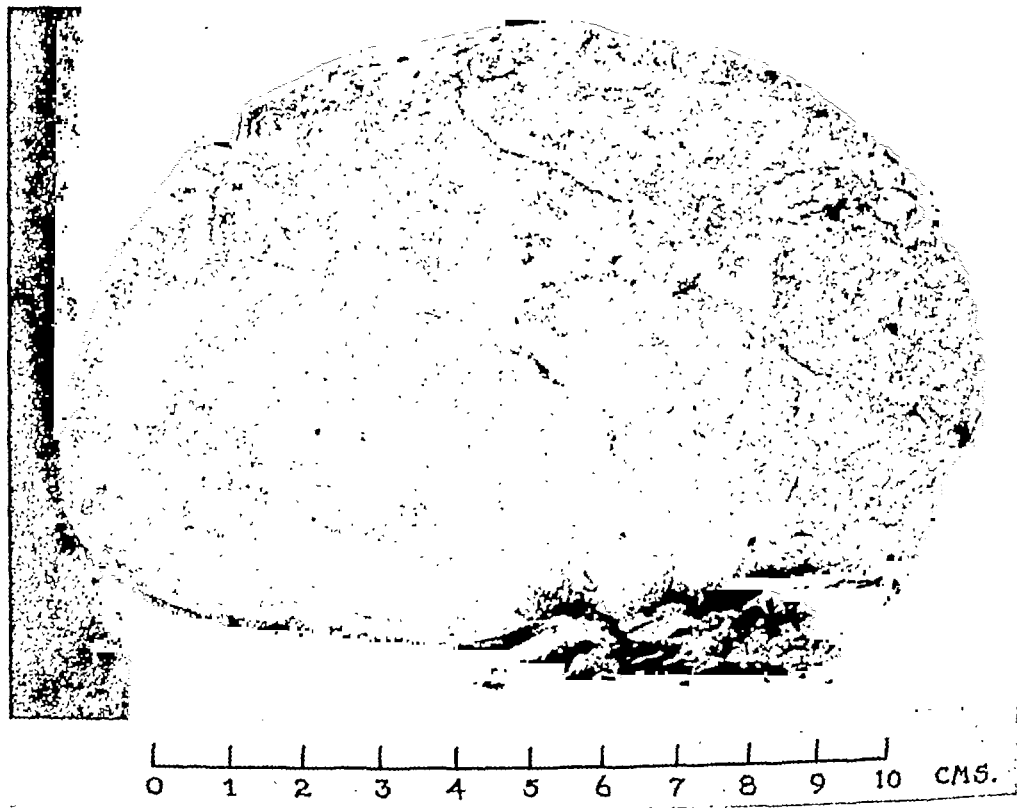


Fig. 4.—Cut surface of the tumor disclosing its glandular appearance. Observe the creamy gray substance interspersed by areas of translucent tissue. The symphysis attaching the tumor to the chest wall can be noted in the lower right-hand corner.

PATHOLOGIC REPORT

Gross Description (Fig. 4).—The specimen consisted of a large tumor mass attached to the pleural surface of a portion of chest wall containing segments of four ribs, each averaging 5 cm. in length, together with the intervening intercostal structures. The tumor was slightly bosselated, ovoid, pink, encapsulated, 14 by 12 by 8 cm., and weighed 1,000 Gm. Cut sections revealed a firm grayish-orange tissue interspersed by small, irregular, rather translucent, myxomatous areas. Thin-walled blood vessels were prominent on the surface of the tumor.

Microscopic Appearance (Fig. 5).—The capsule surrounding the tumor was composed of moderately dense fibrous tissue and was intimately attached to the parietal pleura.

The greater portion of the tumor tissue was comprised of clumps of large, ovoid cells, 15 to 60 μ in diameter, separated into lobules by delicate trabeculate without definite pattern. The cytoplasm of the cells was eosinophilic and contained a varying number of large and

pseudolipoma, lipoblastoma, atypical lipoma, and granular cell myoblastomas may be identical with the typical hibernoma.^{16, 22, 24}

Positive identification of an intrathoracic lipoma can usually be accomplished only by exploratory thoracotomy or post mortem examination. Diagnostic needle biopsy of intrathoracic masses should not be employed as long as hope for curative therapy exists.

CONCLUSION

1. The successful resection of an unusual type lipoma, "hibernoma," occurring within the chest is recorded.
2. The importance of exploratory thoracotomy in the diagnosis of intrathoracic disease is emphasized.

REFERENCES

1. Abbot, A. C., and Webb, W. G. S.: A Case of Intrathoracic Lipoma, *Canad. M. A. J.* 33: 660-661, 1935.
2. Andrus, W. DeW., and Heuer, G. J.: Surgical Treatment of Tumors of the Mediastinum, *Surg., Gynec. & Obst.* 63: 469-482, 1936.
3. Blades, B.: Relative Frequency and Site of Predilection of Intrathoracic Tumors, *Am. J. Surg.* 54: 139-148, 1948.
4. Buettner, A.: Ueber Zwerchfellbrueche hinter dem Brustbein und Fettgewebeschwuelste des Zwerchfells, *Arch. f. klin. Chir.* 202: 154-170, 1941.
5. Bradford, M. L., Mahon, H. W., and Grow, J. B.: Mediastinal Cysts and Tumors, *Surg., Gynec. & Obst.* 85: 467-491, 1947.
6. Heuer, G. J., and Andrus, W. DeW.: The Surgery of Mediastinal Tumors, *Am. J. Surg.* 50: 143-224, 1940.
7. McCorkle, R. G., and Koerth, C. J.: Diagnosis of Intrathoracic Tumors, *Texas State J. Med.* 39: 194-197, 1943.
8. McCorkle, R. G., Koerth, C. J., and Donaldson, J. M., Jr.: Thoracic Lipomas, *J. THORACIC SURG.* 0: 568-582, 1940.
9. Oestern, H. F.: Beitrag zur Kenntnis der intrathorakalen Lipoma, *Zentralb. f. Chir.* 72: 591-596, 1947.
10. Piaggio Blanco, R. A., and Sayagues, C.: Lipoma intratorácico del mediastino anterior, *Arch. urug. de med. cir. y especialid.* 19: 146-153, 1941.
11. Swineford, O., Jr., and Harkrader, C. J., Jr.: Intrathoracic Lipoma; a Case Report, *Ann. Int. Med.* 17: 125-129, 1942.
12. Walker, R. M.: Mediastinal Lipoma, *J. THORACIC SURG.* 6: 89-97, 1936.
13. Watson, M. C., and Urban, J. A.: Mediastinal Lipoma; a Case Report, *J. THORACIC SURG.* 13: 16-20, 1944.
14. Yater, W. M., and Lyddane, E. S.: Lipoma of the Mediastinum, *Am. J. M. Sc.* 180: 79-84, 1930.
15. Brines, O. A.: Personal communication.
16. Brines, O. A., and Johnson, M. H.: Hibernoma, a Special Fatty Tumor; Report of a Case, *Am. J. Path.* 25: 467-479, 1949.
17. Beyers, C. F.: A Case of "Subpleural" Lipoma in a Child, *Lancet* 1: 283-284, 1923.
18. Hess, G. H.: Sub-pleural Fibrolipoma; Report of a Case, *Radiology* 6: 525-527, 1926.
19. Vinson, P. P., and Pembleton, W. E.: Lipoma of Left Main Bronchus; Report of a Case and Review of Literature, *Arch. Otolaryng.* 35: 868-870, 1942.
20. Watts, C. F., Clagett, O. T., and McDonald, J. R.: Lipoma of the Bronchus; Discussion of Benign Neoplasms and Report of a Case of Endobronchial Lipoma, *J. THORACIC SURG.* 16: 132-144, 1946.
21. Wiper, T. B., and Miller, J. M.: Intrathoracic Mediastinal Lipoma, *Am. J. Surg.* 66: 90-96, 1944.
22. Dolley, F. S., and Brewer, L. A.: The Diagnosis and Treatment of Primary Intrathoracic Tumors, *J. A. M. A.* 121: 1130-1136, 1943.
23. Perkins, C. W., and Bowers, R. F.: Liposarcoma of the Mediastinum and Lung, *Am. J. Roentgenol.* 42: 341-344, 1939.
24. Merkel, H.: Ueber ein Pseudolipom der Mamma. (Eigenartiger Fettzellentumor), *Beitr. z. path. Anat. u. z. allg. Path.* 39: 152-157, 1906.
25. Bonnel, F.: Tumeur du creux de l'aisselle, *Bull. et mém. Soc. anat. de Paris* 89: 110-112, 1914-1919.

TABLE I

	LIPOMA (PER. CENT)	HIBERNOMA (PER. CENT)
Naturally occurring conjugated diene fatty acids	1.47	1.06
Naturally occurring conjugated triene fatty acids	0.01	0.01
Naturally occurring conjugated tetraene fatty acids	nil	nil
Linoleic fatty acids	2.04	4.62
Linolenic fatty acids	0.20	0.16
Arachidonic fatty acids	0.11	0.18
Total polyunsaturated fatty acids	3.83	6.03
Oleic fatty acids	50.38	51.06
Saturated fatty acids	45.79	42.91
	100.00	100.00

DISCUSSION

Most intrathoracic lipomas originate from the mediastinum. Hourglass or dumbbell shaped tumors, in their relation to the intercostal space, are less common while bronchial and parietally situated subpleural lipomas are rare.^{3, 5, 17-21} With the increasing incidence of routine and survey chest roentgenograms, most of these tumors will be detected as asymptomatic opacities. Large lipomas will produce signs and symptoms according to their locations. Small tumors will command attention if endobronchially located.

The liability of sarcomatous change thus demanding immediate and complete surgical excision of lipomas has been stressed.^{22,23}

Hibernoma, the particular type of tumor found in this patient, was probably first described by Merkel in 1905, with the term "adenoma of sebaceous gland."²⁴ The word hibernoma was introduced by Gery in 1914 while discussing a case report.²⁵ He believed it to be a neoplasm originating from homologues in man of hibernating glands. The specific form of adipose tissue present in hibernating glands had been known for many years, having been mentioned by Velsch in 1670,²⁶ and later studied by numerous authors.²⁷⁻²⁹ Bonnot³⁰ and Hatai³¹ used the name interseapular gland to depict this structure and reported its occurrence in human beings. From a detailed study of adult and embryonic human tissues, Bonnot traced its origin from the region of the primitive jugular vein. In man the interseapular gland is a paired, light brown, lobulated, irregular, vascular mass, situated on the shoulder and side of the neck between the superior borders of the scapula and clavicle. This glandular structure is composed of lobulated areas of multiloculated or coarsely granular fat cells occasionally surrounding small portions of lymphoid tissue, somewhat resembling the hemolymph glands of lower mammals. A variety of functions has been ascribed to this peculiar tissue: hemopoiesis, hemocatheresis, fat storage, an origin of the autonomic nervous system, part of the thymus, and as an endocrine gland.^{27, 32}

Tumors of this structure have not been recognized except rarely. Brines and Johnson¹⁶ presented the published cases, listing nine as reasonably authentic, seven possible, and describing one new instance. Although they do not approve of the nomenclature, they believe it to constitute a definite oncologic entity. It is probable that some of the neoplasms diagnosed as xantholipoma, myxolipoma,

LATE RESULTS IN FOUR HUNDRED TWENTY TUBERCULOUS PATIENTS SUBJECTED TO THORACOPLASTY

F. HAGN-MEINCKE, M.D.*
ROSKILDE, DENMARK

THE evaluation of the results of thoracoplastic treatment of pulmonary tuberculosis often causes difficulties. Nothing is gained from just stating the percentage of recovery of a small number of patients shortly after the operation. It requires a sufficient number of patients subjected to uniform methods of operation; exact information about this material must be at hand so that the condition of the patients immediately prior to operation is known, and so that it is evident what patients were selected for operation. Furthermore, it requires an observation period of at least two or three years, besides exact criteria, for the judgment of final results. Especially, it must be clear what is required for describing a patient as healthy, in which connection cultivation methods for tubercle bacilli from sputum and gastric lavage water must be considered. A systematic statement of results year by year after the operation would be still better.

The material stated in this paper meets these demands. It involves the total number of thoracoplasty patients in the Øresundshospital, Copenhagen, during the years 1935 to 1941. Altogether, the material comprises 424 patients with pulmonary tuberculosis subjected to paravertebral thoracoplasty on modern principles, including extensive resection of the upper ribs. However, operation in cases of empyema cavities, as well as other atypical operations, have been excluded. The subsequent investigation has given exact information about all the patients during at least three years after the operation: 141 have died, 132 of tuberculosis, 9 of another intercurrent disease, suicide, or the like. Of the latter, 4 died shortly after the operation, so that the permanent result could not be estimated. They have therefore been struck out of the material. The remaining 5 did not die until long after the operation, and as their condition immediately prior to death was known, they have been registered in accordance with their state of recovery in the period prior to death. Accordingly, the material thus corrected will include altogether 420 patients.

Table I shows the distribution according to sex and age. There is a slight preponderance of female patients. The majority of patients were between 20 and 40 years of age, only a very few above 50 and none below 15 years.

Table II shows the duration of the disease prior to operation. As it is impossible to establish the exact moment of onset of the disease, its inception is assumed to be the moment when tubercle bacilli were demonstrated for the first time in the respective patient. The majority of cases were chronic, with a case history of more than two years. Some patients, however, were operated on within six months. Still, that does not mean that fresh cases were submitted

From the Øresundshospital, Copenhagen, Senior Physician, K. Tørring, M.D.

Received for publication Nov. 14, 1949.

*Chief of the Tuberculosis Dispensary.

26. Rasmussen, A. T.: The So-called Hibernating Gland, *J. Morphol.* 38: 147-205, 1923.
27. Rasmussen, A. T.: The Glandular Status of Brown Multilobular Adipose Tissue, *Endocrinology* 6: 760-770, 1922.
28. Shattock, S. G.: On Normal Tumour-like Formations of Fat in Man and the Lower Animals, *Proc. Roy. Soc. Med.* 2: 207-270, 1909.
29. Sheldon, E. F.: The So-called Hibernating Gland in Mammals: a Form of Adipose Tissue, *Anat. Rec.* 28: 331-347, 1924.
30. Bonnot, E.: The Interscapular Gland, *J. Anat. & Physiol.* 43: 43-58, 1908.
31. Hatai, S.: On the Presence in Human Embryos of an Interscapular Gland Corresponding to the So-called Hibernating Gland of Lower Mammals, *Anat. Anz.* 21: 369-373, 1902.
32. Inglis, K.: The So-called Interscapular Gland and Tumors Arising Therein, *J. Anat.* 61: 452-466, 1927.
33. Mosto, D., and Radice, J. C.: Hibernoma de Gery, *Rev. med. y cien. afines*, Buenos Aires 4: 61-68, 1942.
34. Narr, F. C., and Wells, A. H.: Intrathoracic Myxolipoma, *Am. J. Cancer* 18: 912-918, 1933.

Thus, 87 per cent of the cases were cavernous, whereas cavities could not be demonstrated with certainty in 13 per cent (doubtful ring shadows and clearings of less than 1 cm. in diameter were omitted, and systematic tomography was not performed).

The cavities were divided into four groups:

1. Apical cavities, situated in a frontal picture on a horizontal plane through the first costal cartilage
2. Infraclavicular cavities, reaching further downwards, on a horizontal plane through the second costal cartilage
3. Middle zone cavities, at a lower level, on a horizontal plane separating the middle and lower one-third of the lung area
4. Basal cavities in the lower one-third of the lung area

The distribution is seen in Fig. 1.

Fifteen per cent had multiple cavities.

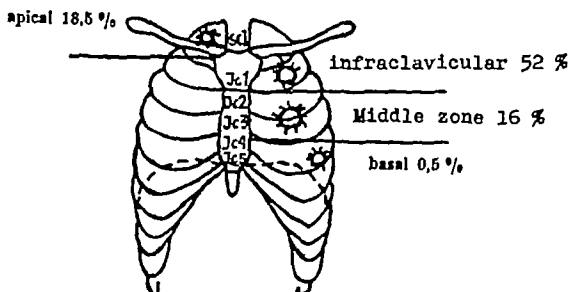


Fig. 1.—Site of the cavities. (Thirteen per cent without cavities.)

Table IV gives an idea of the degree of infiltration: the total affected area in no less than 63 per cent of the cases had an extent corresponding to more than one-half the area of the lung.

The size of the main cavity is seen in Table V. This added to earlier information shows that the material was heavily weighted.

TABLE V. THE SIZE OF THE CAVITIES

	CASES	PER CENT
Small, diameter less than 3 cm.	133	36
Large, diameter 3 to 6 cm.	179	49
Giant cavities, diameter more than 6 cm.	53	15

The next problem is the condition of the contralateral lung, which is seen in Tables VI and VII.

It appears that there was no hesitation in operating on patients with slight contralateral processes, whereas some reserve was shown when the latter extended over more than one-third of the area of the lung. In regard to activity it will be seen that the indications were fairly liberal; 20 per cent had doubtful and no less than 10 per cent clearly active processes.

TABLE I. SEX AND AGE

AGE (YR.)	MALE	FEMALE	ALTOGETHER
Below 15	0	0	0
15 to 20	13	23	36
20 to 25	33	47	80
25 to 30	43	72	115
30 to 35	42	49	91
35 to 40	26	36	62
40 to 45	14	9	23
45 to 50	5	4	9
Above 50	1	3	4
Total	177	243	420

to operation, as part of these patients were actually chronic cases which were not disclosed and diagnosed until at an advanced stage of the disease. The general rule is that pneumothorax is first tried and, if that fails, the patient is confined to bed some months to see whether the processes present signs of regression under conservative treatment.

TABLE II. THE DURATION OF THE DISEASE FROM FIRST DEMONSTRATION OF TUBERCLE BACILLI TO OPERATION

More than 2 years	221
1 to 2 years	57
$\frac{1}{2}$ to 1 year	69
less than $\frac{1}{2}$ year	73
Total	420

Previously applied active treatment is evident from Table III.

The status of the patients immediately prior to operation was as follows:

All were found to be bacillary, most of them by direct microscopy of sputum, a few by cultivation of sputum or of gastric lavage fluid.

Of the greatest importance are the roentgenologic conditions. First the operation side shall be discussed.

TABLE III. ANTECEDENT ACTIVE TREATMENT

	CASES
Pneumothorax impossible	198
Unsuccessful pneumothorax treatment	193
Pneumothorax treatment normally carried through before	29
Phrenic evulsion	15
Paraffin plombage	2
Unsuccessful attempt at extrapleural pneumothorax	5

TABLE IV. THE OPERATION SIDE

SITE OF THE CAVITIES	NUMBER	MULTIPLE CAVITIES	INVOLVEMENT OF MORE THAN ONE-HALF LUNG
Apical	78	7	28
Infraclavicular	218	30	141
Middle zone	67	19	55
Basal	2	0	2
Without cavities	55		37
Total	420	56	263

boldly. The definition of the notion "good chronics" varies somewhat in the reports of the different authors who apply it. In this material a good chronic means a patient who corresponds to the following criteria:

1. Normal temperature during the last two preoperative months
2. Stationary or increasing weight during the same period
3. No severe dyspnea or other more serious complications
4. Age between 15 and 50 years
5. Roentgenogram of lungs shows typical chronic process with retraction (possibly of unknown duration) or nontypical process, but unchanged picture during at least six months; processes involving less than one-third of the area of the contralateral lung, and inactive

TABLE IX. GOOD CHRONICS

	CASES	PER CENT
Good chronics	128	30
Remaining patients	292	
Total	420	

Table IX shows that only 30 per cent of the 420 patients were good chronics; thus, the indications have been very liberal.

The technical details of the operation itself will not be stated here; it is only emphasized that all cases deal with modern thoracoplastic operations involving extensive resection of the upper ribs.

In 261 out of the 420 cases apicolysis was performed in connection with the operation, so that the latter was carried out as a Semb apicolysis thoracoplasty.

TABLE X. THE OPERATION

RIBS	LESS THAN SIX	SIX TO EIGHT	MORE THAN EIGHT	ALTOGETHER
One stage	84	29	2	115
Two stages	12	213	17	242
Three stages	0	21	29	50
Four stages	0	4	9	13
Total	96	267	57	420

Further information is given in Table X, which also shows the extensive division of the operation into stages, although some of the minor apical thoracoplasties were performed in one stage.

Table XI shows the operation mortality (death within two months of the operation). It increases according to the extension of the tuberculous process.

TABLE XI. THE OPERATION MORTALITY

GROUP	NUMBER IN GROUP	OPERATION MORTALITY	
		NUMBER	PER CENT
1	292	15	7
2	102	14	9
3	26	3	12
4	10	4	40
Total	420	36	8.6

TABLE I. SEX AND AGE

AGE (YR.)	MALE	FEMALE	ALTOGETHER
Below 15	0	0	0
15 to 20	13	23	36
20 to 25	33	47	80
25 to 30	43	72	115
30 to 35	42	49	91
35 to 40	26	36	62
40 to 45	14	9	23
45 to 50	5	4	9
Above 50	1	3	4
Total	177	243	420

to operation, as part of these patients were actually chronic cases which were not disclosed and diagnosed until at an advanced stage of the disease. The general rule is that pneumothorax is first tried and, if that fails, the patient is confined to bed some months to see whether the processes present signs of regression under conservative treatment.

TABLE II. THE DURATION OF THE DISEASE FROM FIRST DEMONSTRATION OF TUBERCLE BACILLI TO OPERATION

More than 2 years	221
1 to 2 years	57
$\frac{1}{2}$ to 1 year	69
less than $\frac{1}{2}$ year	73
Total	420

Previously applied active treatment is evident from Table III.

The status of the patients immediately prior to operation was as follows:

All were found to be bacillary, most of them by direct microscopy of sputum, a few by cultivation of sputum or of gastric lavage fluid.

Of the greatest importance are the roentgenologic conditions. First the operation side shall be discussed.

TABLE III. ANTECEDENT ACTIVE TREATMENT

	CASES
Pneumothorax impossible	198
Unsuccessful pneumothorax treatment	193
Pneumothorax treatment normally carried through before	29
Phrenic evulsion	15
Paraffin plombage	2
Unsuccessful attempt at extrapleural pneumothorax	5

TABLE IV. THE OPERATION SIDE

SITE OF THE CAVITIES	NUMBER	MULTIPLE CAVITIES	INVOLVEMENT OF MORE THAN ONE-HALF LUNG
Apical	78	7	28
Infraclavicular	218	30	141
Middle zone	67	19	55
Basal	2	0	2
Without cavities	55		37
Total	420	56	263

TABLE XIII. PERMANENT RESULTS OBTAINED FOR THE GOOD CHRONICS

	NUMBER	PER CENT
Healthy	75	58.6
Ill	32	25.0
Dead	21	16.4
Total	128	

Of the surviving patients 70 per cent healthy

Operation mortality, 3.1 per cent

This is further illustrated in Table XIII. It appears that in the particularly appropriate cases the results are considerably better, with a recovery percentage of 58.6, and the operation mortality has decreased to 3.1 per cent. Besides, at the evaluation of results, it is important to emphasize the rigorous conditions required with regard to the demonstration of tubercle bacilli.

In most foreign materials direct microscopy for tubercle bacilli is the normal procedure, whereas cultivation methods are not used to a very large extent. Exact information about these facts is necessary for estimation of the final results of the operation.

From this material it appears that if cultivation from sputum and gastric lavage water is left out of consideration, 7 per cent more patients will be registered as abacillary, which means that the percentage of recovery will exceed fifty. Exact information about the total material as well as about the good chronics is given in Fig. 2.

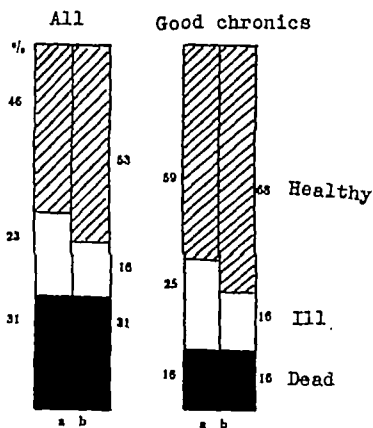


Fig. 2.—Late results: a, with cultivation methods; b, with only direct microscopy for tubercle bacilli.

The results are further illustrated in Tables XIV and XV, which show the importance of the cavity. It is remarkable that the results are very good for the middle-zone cavities, as well as for the large cavities with a diameter of more than 3 cm.

The permanent results of the operations can only be estimated on the basis of a long observation time. It is of no use merely to report—as is done by many authors—that the surgical treatment of cavernous phthisis has afforded this or that result on the patient's discharge from hospital. Despite well-being and apparent collapse of the lung many of these patients continue for a long time to discharge bacilli.

Therefore, the re-examinations of the patients in this study were carried through with an observation time of at least three years, even up to seven years, for the oldest patients. In spite of the difficulties of war all the patients could be traced and exact information about their condition could be obtained, so that the re-examinations could be terminated with a result of 100 per cent.

With regard to the permanent results the patients were divided into three groups: healthy, ill, and dead. To be characterized as healthy the patient must be:

1. Roentgenologically stationary, without signs of residual cavities; without signs of fresh processes or of progression

2. Abacillary, that is,

- (a) in case of expectoration, cultivation of the sputum must be without tubercle bacilli

- (b) if expectoration is impossible and gastric lavage has been performed, cultivation of the fluid must show no growth of tubercle bacilli

3. Without serious complications, such as severe dyspnea, persistent thorax fistulas, etc.

All the other surviving patients were called ill.

Thus, it is seen that cultivation for tubercle bacilli has been performed for all the patients with expectoration. In 75 per cent of the other cases gastric lavage was performed. Thus, it is evident that very rigorous conditions must be fulfilled before a patient is described as healthy, seeing that the mere growth of one colony of tubercle bacilli by cultivation of gastric lavage fluid makes that designation impossible.

TABLE XII. PERMANENT RESULTS OBTAINED IN THE TOTAL BODY OF PATIENTS

	NUMBER	PER CENT
Healthy	193	46.0
Ill	95	22.6
Dead	132	31.4
Total	420	

Of the surviving patients 67 per cent healthy.

Operation mortality, 8.6 per cent

Table XII shows the permanent results obtained in the total material of patients. These results may not seem very brilliant, but it must be borne in mind that clinically as well as roentgenologically the material was heavily weighted, and all types of thoracoplasty were performed, from minor apical operations to the major total thoracoplasties comprising no less than 13 per cent of the operations.

In Table XIX are seen the results of the different types of thoracoplasty, the material being divided according to the form of thoracoplasty planned for each individual patient: (1) apical thoracoplasty resection of up to five ribs; (2) upper lobe thoracoplasty, six to eight ribs; (3) subtotal and total thoracoplasty, above eight ribs.

TABLE XIX. RESULTS OF DIFFERENT OPERATION TYPES

THORACO- PLASTY	TOTAL MATERIAL			GOOD CHRONICS		
	NUMBER	OPERATION MORTALITY (PER CENT)	HEALTHY (PER CENT)	NUMBER	OPERATION MORTALITY (PER CENT)	HEALTHY (PER CENT)
Apical	70	5.7	60	23	0	78
Upper lobe	281	8.0	45	80	3.4	57
Total	60	10.1	35	16	6.3	38
	420			128		

Real understanding of the results of thoracoplasty can only be gained through a comparison with the results obtained in corresponding cases through ordinary conservative treatment.

Such comparisons, however, require a material of conservatively treated patients corresponding to the existing thoracoplastic material.

A material of that description has already been published by Cold (of the Øresundshospital). His material comprises 695 patients with cavernous pulmonary tuberculosis, conservatively treated, and observed through a period of six years. A comparison between the thoracoplastic cases and Cold's older chronic cases shows, as illustrated in Table XX, that apart from some uncertain factors (different observation periods, etc.) the results for patients subjected to thoracoplasty are considerably better than for conservatively treated patients (46 per cent, as against 5.7 per cent).

TABLE XX. CONSERVATIVE AND THORACOPLASTIC TREATMENT

GROUP	NUMBER	HEALTHY (PER CENT)
1. Conservative	31	10
Thoracoplasty	222	53
2. Conservative	52	12
Thoracoplasty	102	42
3. Conservative	102	0
Thoracoplasty	26	31
4. Conservative	72	3
Thoracoplasty	10	0
Total Conservative	264	5.7
Thoracoplasty	420	46.0

This superiority on the part of thoracoplastic treatment applies to Groups 1, 2, and 3, as the graphic description of Fig. 3 will show.

Hardly any good results were registered for the bilateral cavernous cases, through either of the two treatments.

A still better basis of comparison is a group of patients collected by Cold and belonging to Groups 1 and 2, all of whom were fit for thoracoplasty.

TABLE XIV. THE SITE OF THE CAVITIES

	APICAL	INFRACLAVICULAR	MIDDLE-ZONE	BASAL
Healthy	41 (53%)	93 (42%)	27 (41%)	1 (50%)
Ill	15	50	16	1
Dead	21	77	23	0
Total	77	220	66	2

TABLE XV. THE SIZE OF THE CAVITIES

	SMALL	LARGE	GIANT
Healthy	74 (56%)	77 (43%)	11 (21%)
Ill	23	43	16
Dead	36	59	26
Total	133	179	53

The importance of the contralateral lung is seen in Tables XVI and XVII. The best results are obtained when the contralateral lung is sound; however, even in cases of extension of the processes under one-third of the lung area the recovery percentage is good.

TABLE XVI. THE CONTRALATERAL LUNG: EXTENSION

	SOUND	LESS THAN ONE-THIRD	MORE THAN ONE-THIRD
Healthy	67 (55%)	115 (45%)	11 (28%)
Ill	24	64	7
Dead	31	79	22
Total	122	258	40

If the extension of the processes exceeds this limit, or if there are active processes in the contralateral lung, the chances of recovery diminish considerably.

TABLE XVII. THE CONTRALATERAL LUNG: ACTIVITY

	INACTIVE			DOUBTFUL	ACTIVE
	SOUND	SLIGHT CALCIFICATIONS	ACTUAL INFILTRATIONS		
Healthy	67 (55%)	25 (57%)	57 (44%)	38 (45%)	6 (15%)
Ill	24	10	37	17	7
Dead	31	9	36	29	27
Total	122	44	130	84	40

The total extension of processes is of great importance in the prognosis, as seen in Table XVIII.

It is remarkable that in Group 3 (extensive bilateral cases) a fairly great number of patients have recovered.

TABLE XVIII. THE EXTENSION OF THE PROCESSES

GROUP	1	2	3	4
Healthy	117 (53%)	68 (42%)	8 (31%)	0
Ill	48	41	5	1
Dead	57	53	13	9
Total	222	162	26	10

Consequently, as sufficient information could be obtained about the patients all through the period of observation, the total material was made up year by year according to the principles earlier mentioned. Variations through the first five years are given in Fig. 4, corresponding variations for good chronics in Fig. 5.

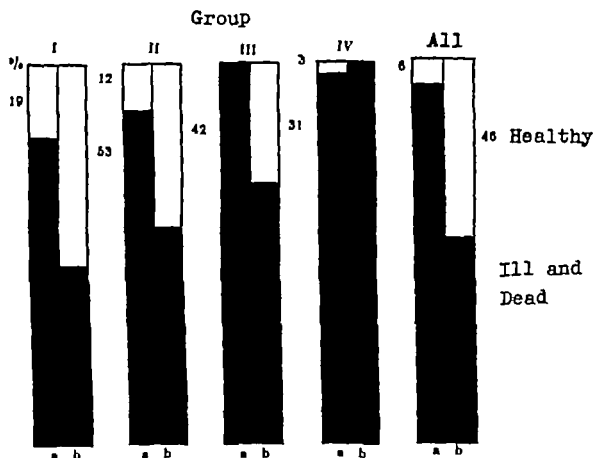


Fig. 2.—Conservative and thoracoplastic treatment. a, Conservative treatment; b, thoracoplasty.

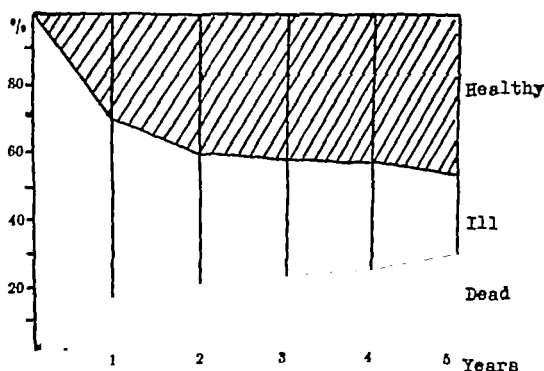


Fig. 4.—Thoracoplasty: 282 cases during five years.

The number of healthy patients shows a great increase during the first two years, after which it is fairly constant. Similarly, the mortality curve rises slightly during the first two years, and only a very little during the following years.

TABLE XXI. GROUPS 1 AND 2

TREATMENT	NUMBER	HEALTHY (PER CENT)
Conservative	166	10
Thoracoplasty	251	51

A selection from the thoracoplastic material of all patients of the first five years in Groups 1 and 2, results in 251 cases, all of them with an observation period of five years. These two materials offer as great correspondence as is practically possible, and show a recovery of 51 per cent, as against 10 per cent in cases of conservative treatment.

TABLE XXII. GROUPS 3 AND 4

GROUP	THORACOPLASTY (FIVE YEARS)			CONSERVATIVE TREATMENT (SIX YEARS)
	3 (PER CENT)	4 (PER CENT)	3 AND 4 (PER CENT)	3 AND 4 (PER CENT)
Healthy	29	0	19	3
Ill	19	20	19	8
Dead	52	80	62	89
Number	21	10	31	396

Conditions regarding Groups 3 and 4 are illustrated in Table XXII. In bilateral cavernous cases results were very poor in both ways of treatment; thoracoplasty, however, may be tried in Group 3 as recovery was, after all, obtained in 29 per cent of the cases, whereas the chances of a conservative treatment are negligible.

Moreover, Cold has made up the results for good chronics, and as the definition of this category within the thoracoplastic material corresponds exactly to the definition used by Cold, the result is a material very suitable for comparison, consisting, as it does, of patients possessing the ideal indications for thoracoplasty.

TABLE XXIII. GOOD CHRONICS

	NUMBER	HEALTHY (PER CENT)
Conservative treatment	39	23
Thoracoplasty	76	62

Table XXIII shows that even if fairly good results are obtained by conservative treatment of this type of patients (23 per cent healthy) thoracoplasty is still very superior to it.

Thus, it follows that within a well-defined group of patients, corresponding to those generally subjected to thoracoplasty, the permanent results of thoracoplastic treatment are found to be far superior to those obtained by conservative treatment.

A fact of very great interest is the examination of the patient's history during the first years after the operation. Only through such an examination is it possible to gain real insight into the future fate of a patient.

If this curve is compared with the corresponding curve of Cold's material (Fig. 9), it is impossible to overlook the striking superiority of thoracoplasty to the conservative treatment in similar cases.

It has often been argued that the effect of thoracoplasty is not so much a lowering of the death rate (at any rate not during the first four to six years), as an increase of the number of permanently abacillary patients, as well as

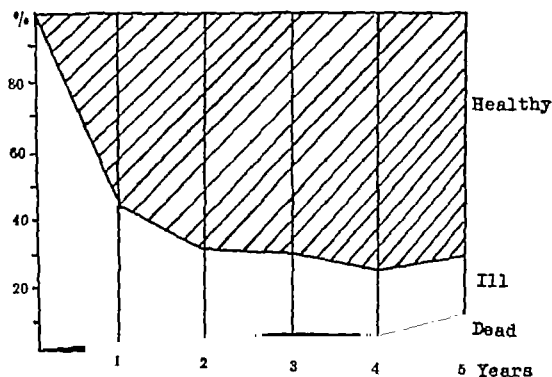


Fig. 7.—Thoracoplasty: 76 good chronics (without cultivation) during five years.

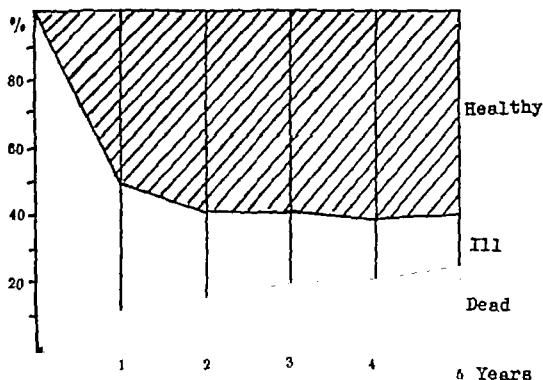


Fig. 8.—Thoracoplasty: 261 cases, Groups 1 and 2, during five years.

quickened debacillation. The curves of this material certainly indicate quicker debacillation, but at the same time the mortality of thoracoplastic cases (Fig. 8) is considerably lower than that of conservative treatment (Fig. 9).

The same thing applies to the good chronics, whose mortality curves are recorded in Fig. 10.

In other words, the permanent results of thoracoplasty cannot be evaluated until after an observation period of at least two years.

In the curve describing good chronics is seen the far greater percentage of recovery, as well as the very low mortality during the five years, within this group of patients.

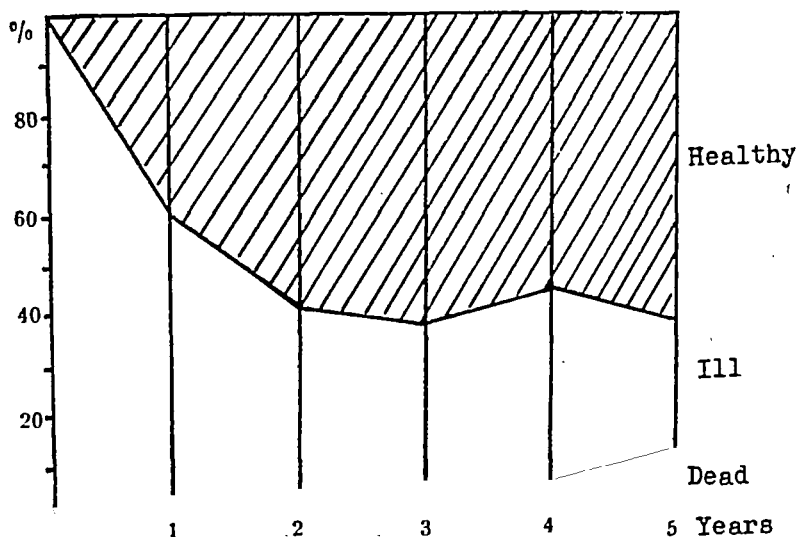


Fig. 5.—Thoracoplasty: 76 good chronics during five years.

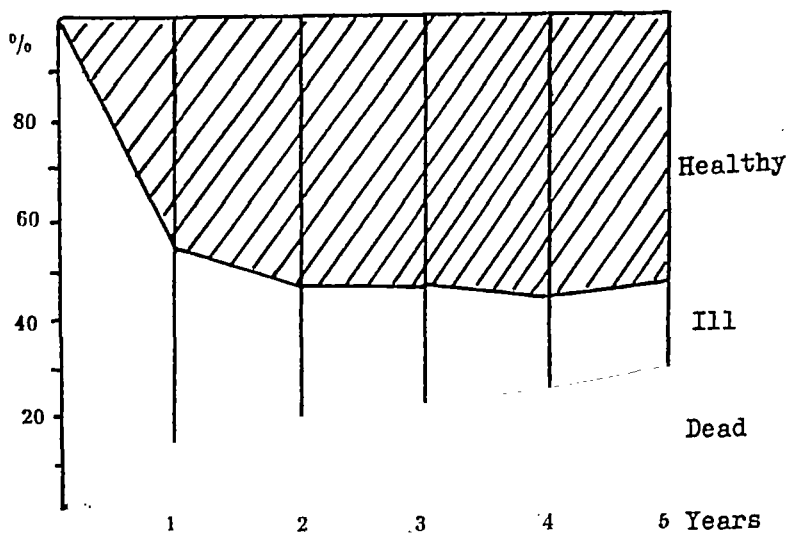


Fig. 6.—Thoracoplasty: 282 cases (without cultivation) during five years.

For comparison with materials in which only direct microscopy for tubercle bacilli is used, Figs. 6 and 7 illustrate the corresponding curves with results without cultivation methods. The results show up to still greater advantage, and already after one year the condition seems constant.

Finally, to point out once more the good results of thoracoplasty compared with those of conservative therapy, the history of Groups 1 and 2 are given through five years, as seen in Fig. 8 (without cultivation methods).

patients were ill. Twenty-seven per cent of these were in good general condition and a roentgenologic examination showed apparently good collapse; no residuary cavities could be demonstrated with the methods used, and the x-ray picture remained stationary.

However, in 10 patients, examination of sputum showed plus tubercle bacilli directly, and, in 8, plus tubercle bacilli on cultivation. The remaining 9 showed plus tubercle bacilli on cultivation from gastric lavage water. In some cases cultivation showed growth of only one, or a few, tubercle bacilli colonies; thus, after all, thoracoplasty caused a not inconsiderable improvement of the general condition. Further research will show the prognosis of such cases.

DISCUSSION

Compared with cases of conservative treatment, this survey has shown fairly good results of the thoracoplastic treatment of chronic cavernous phthisis.

If the operation material is selected from the group of good chronics, excellent results are obtained for all forms of thoracoplasty, namely, recovery of rather more than 60 per cent, as against 20 to 25 per cent by conservative treatment. Further, the operation mortality is very low, about 3 per cent. Thus, within this field thoracoplasty must no doubt be considered the fundamental therapy, and preferable to conservative treatment. On the other hand, the question of the value of thoracoplasty compared with other methods, for example, extrapleural pneumothorax, has not been dealt with in the present work.

Extension of thoracoplasty indications beyond these limits—so that patients with poorer general condition and major contralateral processes are included—will, of course, lower the percentage of recovery. Examinations have shown that even in case of inactive affections in the contralateral lung of up to one-third of the lung area, the results obtained are far better than corresponding results of conservative treatment; in fact, even for doubtfully active processes in the contralateral lung, possibly with pneumothorax, fairly good results are obtained. Similarly, examinations regarding the size of the cavity on the operation side show that good results are obtained even for cavities with a diameter of up to 6 cm. These cases which, with regard to the total extension of the processes, belong to Groups 1 and 2, show a recovery percentage of about 50 and an operation mortality of 6 or 7 per cent, that is, far better than by conservative treatment, which can boast only about 10 per cent healthy patients in corresponding cases. Thus, a considerable extension of thoracoplasty indications beyond the good chronics is fully justified.

The point, then, is where to draw the limit, or, in other words, does thoracoplasty pay in cases of heavy bilateral extension or of bilateral cavernous affection? As mentioned previously, practically no good results are obtained in these groups by conservative treatment. In cases of bilateral cavity, thoracoplasty does not, either, show any good results, even if the material contains few cases of this category, and probably should not, as a rule, be applied to such patients. On the other hand, thoracoplasty of advanced cases without contra-

Thus, it must be admitted that modern thoracoplasty decreases mortality to a large extent compared with conservative methods, at any rate during the first five years after operation.

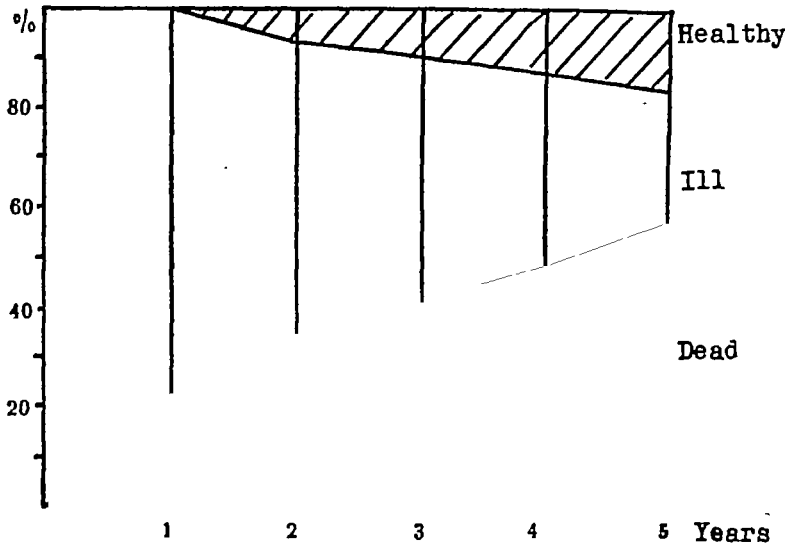


Fig. 9.—Conservative treatment: 90 cases, Groups 1 and 2, during five years.

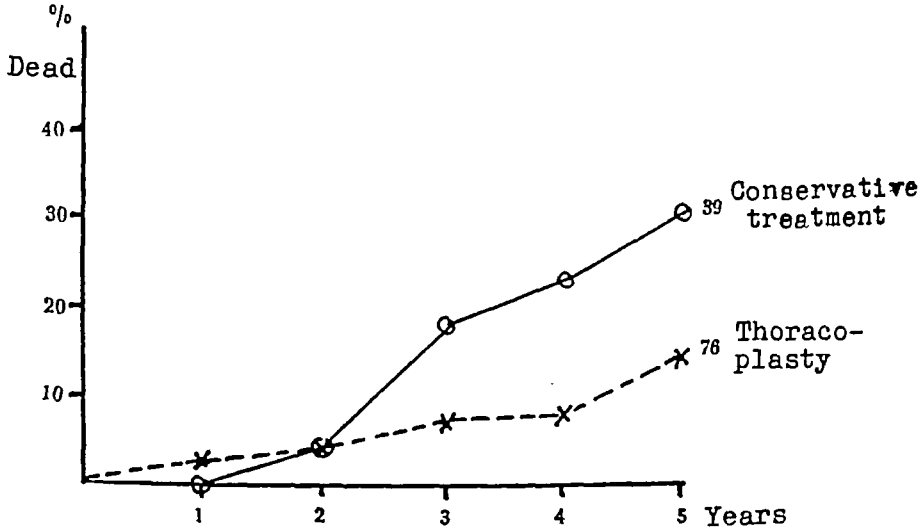


Fig. 10.—Good chronics: mortality curve, one to five years.

It should be remembered, too, that the description of a patient as "ill" does not imply that he is completely an invalid. Part of the patients had a large cavity and plenty of sputum before the operation, and their condition was unstable. The operation caused considerable improvement of the general condition, involving apparent collapse of the cavity, decrease of sputum, etc., the patient feeling well and fit for work; however, examination with cultivation from sputum and gastric lavage water revealed occasional growth of bacilli. Thus, it may be stated from the thoracoplastic material that after five years 63

AN EVALUATION OF APICOLYSIS IN THORACOPLASTY

F. HAAG-MEINCKE, M.D.*

ROSKILDE, DENMARK

DURING the technical development in recent years of thoracoplastic operations for the treatment of pulmonary tuberculosis, the ideal kept in view all along has been adhesionless pneumothorax with its concentric retraction of the lung in all three planes. Through modern selective thoracoplasties involving radical resection of the upper ribs, it is possible to obtain intensive collapse of the upper parts of the lung from side to side and also, to some extent, from front to back. In a great many cases this will be sufficient for a possible cavity to collapse completely; however, in some cases where the apex of the lung adheres upward medially toward the mediastinum, the roentgenogram will sometimes reveal an oblong, vertical residuary cavity. This absence of collapse in apicocaudal direction has led to attempts at combining thoracoplasty with extrapleural loosening of the lung apex so as to bring about retraction in all three directions.

These attempts have led to such operations as paraffin plombage and extrapleural pneumothorax, but also in connection with thoracoplasty apicolysis has earlier been used with little success. Semb, with his extrafascial apicolysis thoracoplasty, was the first to create a really radical and permanent apicolysis. This is the principle of his method: (1) radical and selective apicolysis performed through open dissection in the extrafascial layer; (2) radical rib resection, the extent of which corresponds to the apicolysis or more than that, so as not to leave behind any fragments of ribs that might "draw out" the lung again; (3) mobilization of rib periosteum following the surface of the lung, so that regeneration of the ribs will take place immediately over the loosened, collapsed part of the lung apex. This prevents re-expansion of the lung as well as of lung hernias.

Experience from literature on the subject yields little information as to indications for apicolysis. The use of the method seems to be a matter of routine, at any rate in operations in the upper lung regions. Results seem to be fairly satisfactory, though no reliable basis of comparison with nonapicolysis cases is at hand. It should, in this connection, be remembered that thoracoplastic technique has developed so rapidly in recent years that it has been impossible to form a lasting impression of an operation method until the next was introduced. Evaluation of these methods requires an observation time of at least two or three years after the operation, and if at the same time a comprehensive material is to be provided, it will take some years to collect this material, especially if it must be done in the same hospital.

Attention should therefore be directed to that development of thoracoplasty, including division of the operation into several stages and radical resec-

*Chief of the Tuberculosis Dispensary.

From the Øresundshospital, Copenhagen, Senior Physician, K. Tørning, M.D.

Received for publication Nov. 14, 1949.

lateral cavity has, after all, yielded good results; as mentioned, out of 21 patients, 25 per cent were healthy after five years, and the operation mortality was only 5 per cent. This means that thoracoplasty may afford some chance of recovery if special indications speak in favor of it, for example, if the patient insists on active measures because he is heavily affected mentally at the prospect of a prolonged and hopeless conservative treatment.

The summing-up of the thoracoplastic material, moreover, shows that the good results are obtained at an early stage, appearing as they do, within the first two years after the operation, after which time the condition remains fairly constant, that is, quick debacillation and permanent increase of the number of abacillary patients. Thus, modern thoracoplasty of chronic cavernous phthisis has shown its superiority to conservative therapy, even in case of widely extended indications, and should occupy a dominating position within the treatment of these forms of tuberculosis.

REFERENCES

- Cold, S.: The Prognosis of Pulmonary Tuberculosis by Conservative Treatment, Copenhagen, 1935 (Danish, with an English summary).
Semb, C.: Thoracoplasty With Extrafacial Apicolysis, *Acta chir. Scandinav.* (Supp. 37) 76: 1-85, 1935.

two groups are, perhaps, not after all quite comparable with each other; thus the apicolysis group might contain some more poor objects of operation, who lower the recovery percentage as much as apicolysis might increase it. Apart from the fact that examination of the material reveals no sign of abnormal distribution, a somewhat closer analysis of results will be necessary.

In order to obtain two uniform groups of patients it would be practical to select the good chronics mentioned in the preceding article,* who fulfill the ideal indications for thoracoplasty. Such a selection would eliminate all poor objects of operation, who might otherwise be unevenly distributed, and provide two easily comparable groups. Examination of the material according to the previously mentioned criteria for the designation of good chronics shows that there are altogether 76 cases of that description. Table II shows the results of the two groups: 31 without and 45 with apicolysis.

TABLE II. SEVENTY-SIX GOOD CHRONICS

	WITHOUT APICOLYSIS	WITH APICOLYSIS
Healthy	20 (64.5%)	27 (60.0%)
Ill	7	11
Dead	4	7
Total	31	45

This statement, too, does not show any deviations in favor of the use of apicolysis.

However, further elucidation of the question is possible through division of the material according to the extension of the lung processes, as they appear on the roentgenogram. The following table shows the results of the different groups with and without apicolysis.

TABLE III. EXTENSION OF PROCESSES

GROUP		WITHOUT APICOLYSIS	WITH APICOLYSIS
1	Healthy	32 (53%)	47 (58%)
	Ill	14	18
	Dead	14	10
2	Healthy	27 (48%)	22 (42%)
	Ill	13	13
	Dead	17	18
3	Healthy	4 (33%)	2 (22%)
	Ill	2	2
	Dead	6	5
4	Healthy	0	0
	Ill	0	1
	Dead	5	4

Comparison shows that only in Group 1, the mildest cases, the percentage of "healthy" patients treated with apicolysis is slightly higher than that of the nonapicolysis group. This difference, however, is negligible, and also in the other groups with larger extensions of the processes apicolysis has had no favorable effect.

On the other hand, great importance must be attached not only to the extension of processes, but also to the site of possible cavities; and so it will be

*See page 837 for article.

tion of the upper ribs, which took place immediately before, or simultaneously with, the first appearance of apicolysis. The improvement of results appearing from articles on the subject, may be due to these circumstances as well as to apicolysis, and it is hard to decide which of these is more responsible for it. It should not be left unnoticed that operators who do not make use of apicolysis, in many cases obtain results equal to those of Semb.

It is evident from this that the value of apicolysis has not yet been established, owing to the absence of a material suitable for thorough comparison. In the following, attempt is made to provide such a material from among the thoracoplasty patients of the Øresundshospital during the years 1935 to 1941. Here, K. Tørning, as one of the first operators outside Norway, has subjected this method to examination. At first apicolysis was used only as an experiment, and then only in case of apical medial cavities; later on, however, it was used more extensively. All through this period, in which, incidentally, the operative technique used was on the whole the same, there was a suitable number of operations with, as well as without, apicolysis. As early as 1940, Tørning expressed some scepticism toward the use of apicolysis in ordinary upper-lobe thoracoplasty, whereas he did not question its value in apical operations.

The material to be used for a comparative investigation was described in detail in the preceding article, "Late Results in 420 Tuberculous Patients Subjected to Thoracoplasty,"* and the patients of the years 1935 to 1939 comprise altogether 282 nonselective cases of lung tuberculosis treated with thoracoplasty. The material is evenly distributed, apicolysis being performed in connection with thoracoplasty in 148 cases, while the remaining 134 patients were not submitted to apicolysis. The results of these thoracoplasties were divided according to the principles mentioned earlier into three categories: healthy, ill, and dead. The same observation period, five years after operation, applies to all cases. This should provide a good basis for examining the value of apicolysis.

Table I gives a statement of the total material after an observation period of five years.

TABLE I. PERMANENT RESULTS AFTER FIVE YEARS

	134 WITHOUT APICOLYSIS		148 WITH APICOLYSIS	
	NUMBER	PER CENT	NUMBER	PER CENT
Healthy	63	47.0	71	48.0
Ill	29	21.6	34	23.0
Dead	42	31.4	43	29.0
Operation mortality	11	8.2	11	7.4

It appears from this statement that the figures of the two groups correspond exactly to one another, which means that at any rate in the total material apicolysis does not seem to have carried any effect in one direction or the other. However, the statement does not necessarily signify that apicolysis is of no value at all. In a limited number of cases it may very well have a certain favorable effect, which, on the other hand, is too slight to affect the total material. Besides, various factors may intervene, as in cases when the

*See page 837 for article.

attempted to single out such cases and eliminate all patients with multiple cavities if these were situated outside the field of the largest cavity. For this reason, altogether 22 cases had to be left out of consideration. The corrected figures, however, show a percentile accordance with the figures of the tables quoted, so that this circumstance does not affect the results.

Next it should be mentioned that in grouping the cavities, one extending over several of the lung fields mentioned is here described as belonging to the lowest field it touches, that is, that a cavity described as infraclavicular may very well extend into the apex area, but not into the middle-zone area; on the other hand, an apical cavity is situated exclusively in the apex. In other words, besides the apical cavities discussed previously, there is an additional number touching the apex. In this material there are, besides the 54 purely apical cavities, 39 infraclavicular cavities extending into the apex, and, finally, to these may be added 3 cases of large cavities extending from the middle zone right into the apex. An illustration of this is given in Table VII. It appears that there is only a slight difference in favor of cases treated with apicolysis. Correction with regard to multiple cavities does not alter the picture.

TABLE VI. APICAL CAVITIES

GROUP	WITHOUT APICOLYSIS		WITH APICOLYSIS	
	NUMBER	HEALTHY (PER CENT)	NUMBER	HEALTHY (PER CENT)
1	5	20	29	73
2	8	50	10	30
3	0	0	0	0
4	1	0	1	0

In order to see clearly the effect of apicolysis in apical cases it might, besides, be useful to examine the cases which only showed apical cavities and, as before, distinguish between medial and lateral cavities. This is shown in Table VIII, in which are stated the 54 cases of apical cavities; however, cases of multiple cavities all through the area were not considered; thus, only 43 cases of solitary cavities were registered. Distinction was made between medial cavities whose medial edges reach as far as, or into, the processus transversus area, and the remaining lateral cavities.

TABLE VII. CASES OF CAVITIES TOUCHING THE APEX

	WITHOUT APICOLYSIS	WITH APICOLYSIS
Healthy	9 (83%)	29 (42%)
Ill	7	19
Dead	11	21
Total	27	69

No great effect seems traceable from the use of apicolysis either with regard to medial or to lateral cavities.

Accordingly, this examination of the importance of apicolysis in proportion to the extension of processes and the site of cavities reveals that in the more severe cases of infraclavicular and middle-zone cavities which comprise the

useful to examine this circumstance in order to find out, if possible, whether in cases of uniform extension apicolysis has proved valuable to any particular group of patients.

For that purpose analysis was made with a view to the site of the cavity, and to the results of groups with and without apicolysis. It appears that the present material contains altogether 239 undoubtedly cavernous cases. Division according to the site of the cavity was made into the four groups mentioned earlier, namely, apical, infraclavicular, middle-zone, and basal cavities; in case of multiple cavities only the largest was considered. The distribution shows 54 cases of apical cavity, 137 of infraclavicular, 46 of middle-zone, and only 2 cases of basal cavity. Disregarding the 2 cases of basal cavity, the results of the different groups are stated in Table IV.

TABLE IV. SITE OF THE CAVITY

	APICAL		INFRACLAVICULAR		MIDDLE-ZONE	
	WITHOUT APICOLYSIS	WITH APICOLYSIS	WITHOUT APICOLYSIS	WITH APICOLYSIS	WITHOUT APICOLYSIS	WITH APICOLYSIS
Healthy	5 (36%)	18 (45%)	31 (47%)	33 (47%)	15 (50%)	7 (44%)
Ill	4	14	12	13	6	4
Dead	5	8	23	25	9	5
Total	14	40	66	71	30	16
	54		137		46	

Examination of these figures shows no effect of apicolysis when applied to middle-zone and infraclavicular cavities; in the group of apical cavities, however, the number of healthy patients is slightly greater among apicolysis cases, though the difference is not significant. A combination of this division with a division according to the extension of processes, as shown in Tables V and VI, yields the same result for infraclavicular and middle-zone cavities; in groups of uniform extension and the same cavity position there is no sign that apicolysis is effective. On the other hand, with regard to apical cavities Group 1 shows a higher percentage of recovery for apicolysis cases, namely, 73 per cent healthy as against 20 per cent in the nonapicolysis group. The figures of the latter group, however, are so low that care should be taken not to draw too far-reaching conclusions. Besides, some remarks must be added to the analysis stated. In the first place, in cases of multiple cavities only the largest cavities were taken into consideration. This might affect the result, as there are some cases of cavities scattered over a large part of the lung area. Accordingly, it was

TABLE V. EXTENSION AND CAVITY POSITION

GROUP	INFRACLAVICULAR		MIDDLE-ZONE	
	66 WITHOUT APICOLYSIS (PER CENT HEALTHY)	71 WITH APICOLYSIS (PER CENT HEALTHY)	30 WITHOUT APICOLYSIS (PER CENT HEALTHY)	16 WITH APICOLYSIS (PER CENT HEALTHY)
1	64	61	50	50
2	38	38	62	45
3	33	22	33	0
4	0	0	0	0

middle-zone or basal processes in which the apex is not affected and in which, therefore, apicolysis has not been used. The apicolysis cases, on the other hand, usually show processes stretching right from the apex down through the lung area. If correction is tried in these cases it appears that there is no change in favor of apicolysis; however, the figures are fairly small. Thus, the statement shows that only with regard to purely apical operations is there, perhaps, some possibility of effect through the use of apicolysis.

Finally, it may be added that examination of the importance of dividing the operation into several stages shows no indication that apicolysis should have a favorable effect within groups of uniform types of operation performed partly in one, partly in several stages, except in the group comprising apical thoracoplasties performed in one stage. Here the percentage of recovery is higher in apicolysis cases, which is in accordance with the earlier results.

Accordingly, the total result of these examinations shows that the use of apicolysis as a part of thoracoplastic operations has proved unable to affect the permanent results of the total material, and with regard to infraclavicular and lower processes, that is, upper-lobe and total thoracoplasties, which include three-fourths of the operations performed, a closer analysis shows that no effect of apicolysis was traceable. In case of purely apical processes some favorable effect of apicolysis could not with certainty be precluded in the material at hand.

SIZE OF THE APICOLYSIS

After these reflections, which largely reduce the use of apicolysis, the question presents itself whether the apicolyses performed in this material were radical enough. Accordingly the 148 apicolysis cases have been examined with the object of ascertaining the extent to which the lung was loosened. Through the apicolysis the lung, as is known, sinks to at least the level of the fourth rib at the back, and the apex is seen lying free 6 or 8 cm. below the bundle of nerves and vessels. Analysis of the material shows that the loosening of the apex was now and then impeded by extensive adhesions; occasionally reserve was shown for fear of cavity rupture. Therefore, in some cases the apicolysis was not completed, only a partial apicolysis being performed, and, in consequence, distinction has been made between these minor apicolyses and more extensive loosening. Among the former are classed apicolyses in which the loosening does not extend lower than the third rib, whereas the major loosening all extend to the fourth or even lower ribs.

It appears that most of the apicolyses were satisfactorily performed, 123 out of 148 being major apicolyses (83 per cent)—of these no less than 60 per cent as far as about the fifth rib, or lower. The remaining 25 were minor or partial apicolyses.

It might be conceivable that the patients subjected to minor apicolyses fared worse than the rest. However, Table X, in which the results of these groups after five years are stated, shows that this is not so.

After these statements the question might be asked whether a number of drawbacks cling to the use of apicolysis in connection with thoracoplasty, even

majority of cases, apicolysis seems to have had no effect. However, in cases of slight extension and apical cavity it cannot be precluded that apicolysis may have a certain profitable effect.

These questions may also be regarded under a different angle, if the problem is analyzed with a view to the extent of the thoracoplastic operation. In planning this, attention was paid not only to the size and site of the cavity, but the extension of the total process was estimated before decision was taken

TABLE VIII. CASES OF APICAL CAVITIES

	MEDIAL CAVITY		LATERAL CAVITY	
	WITHOUT APICOLYSIS	WITH APICOLYSIS	WITHOUT APICOLYSIS	WITH APICOLYSIS
Healthy	2 (50%)	5 (50%)	3 (43%)	10 (46%)
Ill	1	1	2	8
Dead	1	4	2	4
Total	4	10	7	22
	14		29	

as to how many ribs should be resected. Below, the material is divided, according to the extent of the operation, into the three main groups earlier mentioned: apical thoracoplasty (up to 5 ribs), upper-lobe thoracoplasty (6 to 8 ribs), and subtotal and total thoracoplasties (more than 8 ribs). Next, correction has been made for unsuccessful and incomplete thoracoplasties, as otherwise a number of large operations which could, for several reasons, not be completed, would be described as unsuccessful apical thoracoplasties. Altogether these cases total 55 apical, 185 upper-lobe, and 42 total or subtotal thoracoplasties, and the results of the various groups with and without apicolysis are stated in Table IX.

TABLE IX. SIZE OF THE OPERATION

	APICAL THORACOPLASTY		UPPER-LOBE THORACOPLASTY		TOTAL THORACOPLASTY	
	WITHOUT APICOLYSIS	WITH APICOLYSIS	WITHOUT APICOLYSIS	WITH APICOLYSIS	WITHOUT APICOLYSIS	WITH APICOLYSIS
Healthy	7 (41%)	26 (68%)	41 (47%)	41 (42%)	15 (52%)	4 (32%)
Ill	5	8	19	22	5	4
Dead	5	4	28	34	9	5
Total	17	38	88	97	29	13
	55		185		42	

Examination of these figures shows, on the whole, a repetition of the circumstances applying to cavity position, as after all was to be expected. Apical operations show a somewhat higher percentage of recovery in the apicolysis group, yet the difference is not significant. A division into uniform groups with regard to the extension of processes according to Cold shows similar conditions in Group 1, to which the majority of cases belong. On the other hand, no advantage seems to arise from the use of apicolysis in case of major operations (upper lobe and total thoracoplasty). In this connection it must be remembered, however, that the apicolysis group contains more severe cases than the nonapicolysis group, the latter containing a number of cases with fairly limited

eations in the chest wall occur with the same degree of frequency in the two groups, the difference was due to the more serious, and intractable, subscapular fistulas occurring only in the apicolysis group. It is thus to be feared that apicolysis involves a greater risk of troublesome wound infections. Here it must be borne in mind that the material dates from the time before penicillin and streptomycin came into use in connection with thoracoplasty.

AFFECTION OF THE VASCULAR SYSTEM

Affection of the vascular system involving lower blood pressure and shock-like conditions is of frequent occurrence in thoracoplastic operations. Already during the resection of the first rib, as well as during the apicolysis itself, a lowering of the blood pressure is often noticeable, sometimes transient, but also passing into a surgical shock proper, developing in connection with, or subsequent to, the operation. Unfortunately in the present hospital there have been no examinations of this circumstance, such as determining hemoconcentration, or the like; thus, the only means at hand to diagnose the shock have been the blood pressure, the pulse rate, and the clinical estimate. Accordingly it has also been impossible to ascertain whether this lowering of the blood pressure is at all connected, pathogenically, with actual shock.

In order to overcome these difficulties it was decided, with a view to ascertaining the affection of the vascular system, to examine the circumstances of the blood pressure, as done by Semb in his material, in which the dividing line is drawn at a systolic blood pressure of 85 mm. Hg. It then appeared that a drop below this line occurred in 41 cases without apicolysis, that is, 26 per cent. In the apicolysis cases the drop occurred 54 times, or 21 per cent. Thus, apicolysis has not increased the number of such cases, the less serious of which are, as a rule, controlled by placing the patient's head in a low position and administering ephedrine injections, whereas in all shocklike conditions blood transfusion is the sovereign remedy.

OTHER COMPLICATIONS

Other important complications are postoperative pneumonias. As especially Semb has demonstrated, the development in this respect is an increase of bronchial secretion after the operation and, as a consequence of poor expectoration, the formation of atelectasis and subsequent infection and pneumonia. In other cases secretion may obstruct the bronchi, thereby involving danger of choking. A number of deaths are, no doubt, due to such conditions and not, as generally stated, to weakening of the heart. On the whole, heart complications are rare and not very important in local thoracoplasties. Cases of highly increased bronchial secretion and threatened choking were observed in 6 non-apicolysis cases and in 9 cases treated with apicolysis. In several of these cases bronchoscopy was performed and the secretion was aspirated. Pneumonia was observed in 6 nonapicolysis cases and in 8 apicolysis patients. Thus, the two groups present no very great variation in these respects.

Other complications, which occurred only in a limited number of cases, were, for example, lesions of nerves, vessels, pleural ruptures, cavity ruptures,

TABLE X. SIZE OF THE APICOLYSIS

	MINOR APICOLYSIS	MAJOR APICOLYSIS
Healthy	15 (60%)	56 (46%)
Ill	4	30
Dead	6	37
Total	25	123

if the final results of the groups with, and without, apicolysis show little difference; or, in other words, whether the procedure in question may be the cause of more, or of additional and more serious, operative complications than is the case without the use of apicolysis.

As it is here a question not of late results, but only of the unfavorable complications following immediately upon operation, attempts have been made to ensure as comprehensive a material as possible by basing the examination not only on the patients observed through five years, but on the total material. Altogether there are 420, of whom 261 were subjected to operation with apicolysis, and 159 to operation without apicolysis.

INFECTION IN THE WOUND CAVITY

Examination of the material shows that 75 out of 420, that is, 18 per cent, had postoperative wound infection. As for degree and description of the infection, distinction was made between two groups.

The first group contains cases of slight infection, involving inflammation of the skin, the subcutaneous tissue or the superficial muscles; as a rule there was a subcutaneous undermining, often with fistula formation resulting in purulent secretion, generally of short duration; in a number of cases, however, these fistulas would persist for several months. In cases where the secretion was subjected to a bacteriologic examination it was, as a rule, a matter of ordinary pyogenic bacteria, and in a very few cases, of tuberculous infection. The second group contains more serious cases involving deeper infections in the subscapular cavity with fistula formations which, unlike those of the former group, were mostly very resistant to therapy and often kept on persistently in spite of excisions, lavages, or other operations. In some cases they involved invalidity and had existed for several years. Tuberculous infection occurred in 5 cases, while the remaining cases were due to ordinary pyogenic infection.

Distribution with regard to apicolysis is shown in Table XI.

It appears that the infection percentage was somewhat higher in the apicolysis group, and, more important, that while the slight wound compli-

TABLE XI. WOUND INFECTION

INFECTION	WITHOUT APICOLYSIS (169)		WITH APICOLYSIS (261)	
	NUMBER	PER CENT	NUMBER	PER CENT
In chest wall	25	16	41	16
Subscapular	0		9	3
Total	25	16	50	19

recorded. In 4 cases determinations on the left side were impossible, due in 1 case to a previous pneumonectomy, in the other 3 to tuberculous adhesions. In 1 case a pneumonectomy had been performed on the right, thus preventing determinations being made on that side. The observations were made by a single observer (J. E. H.)

PERICARDIAL SINUSES AND RECESSES

The fibrous covering of the pericardium is attached caudad to the diaphragm, cephalad to the deep cervical fascia, and laterally extends as the adventitial coverings of the great vessels. The parietal layer of serous pericardium lies on the under surface of the fibrous covering, but at the points of both arterial and venous attachments to the heart, it is reflected upon the heart as the visceral layer.

Cephalad, there is a dorsal communication between the right and left sides of the pericardial cavity which is bounded in front by the aorta and pulmonary artery, and behind by the superior vena cava, left atrium, and right pulmonary artery. This is the "transverse sinus" (Fig. 1). Caudalward, another dorsal sinus is present on the posterior aspect of the atria on the left side. It has been termed the "oblique sinus of the pericardium."²¹ It is a blind cul-de-sac bounded posteriorly by the parietal pericardium, anteriorly by the atria, cephalad by the pulmonary veins, and on the right side by the inferior vena cava (Fig. 2).

Many unnamed recesses in the pericardial sac have been described by anatomists. Allison described two of these recesses and designated them as a "left pulmonary recess" and a "postcaval recess." The left pulmonary recess is bound cephalad by the left pulmonary artery, caudad by the left superior pulmonary vein, and medially by the vestigial fold of Marshall (Fig. 3). The postcaval recess is located on the right side, and is bounded anteriorly by the superior vena cava, cephalad by the right pulmonary artery, and caudad by the right superior pulmonary vein (Fig. 4). Another recess to which we would like to refer for descriptive purposes may be termed the "right pulmonary recess," located between the superior and inferior pulmonary veins on the right side (Fig. 4).

THE PULMONARY VESSELS

The most cephalic extension of the parietal layer of serous pericardium was found to be upon the aorta. This occurred on the average about 6 cm. above the origin of the aorta from the heart measured on its anterior surface. From this point, the line of reflection on the left side extends over the anterior and inferior surfaces of the left pulmonary artery (Fig. 2). A little more than one-half the circumference of the artery is easily visible (Table I). The serous pericardium then descends caudad and reflects upon the superior, anterior, and inferior surface of the left superior pulmonary vein, leaving about two-thirds of this vessel's circumference accessible within the sac. Continuing caudalward the serous layer now reflects upon the superior, inferior, and anterior surface of the left inferior pulmonary vein. This vessel is the most palpable, four-fifths of the circumference bulging into the pericardial

INTRAPERICARDIAL ANATOMY IN RELATION TO PNEUMONECTOMY FOR PULMONARY CARCINOMA

JOHN E. HEALEY, JR., M.D., AND JOHN H. GIBBON, JR., M.D.
PHILADELPHIA, PA.

IN 1946, Allison² advocated intrapericardial ligation of the pulmonary vessels in performing pneumonectomy for carcinoma of the lung, in order to permit a safer, and yet more radical, operation. In 44 of 102 patients subjected to pneumonectomy for cancer of the lung in the last three years at Jefferson Hospital, the pulmonary arteries and veins were ligated and divided within the pericardial sac. In some of the 44 patients the pericardium was opened because it was easier and simpler to ligate and divide the great vessels in this situation. In many cases, however, pneumonectomy would not have been possible without using the intrapericardial approach. In large hilar tumors it is difficult, if not impossible, to expose the pulmonary veins outside the pericardium. In carcinomas originating in the lower lobe, the pericardium itself may be directly invaded and must be removed. Not infrequently tumor tissue spreads along the wall of the inferior pulmonary vein, or grows in its lumen, completely occluding it. In all such cases it is impossible to remove the growth in its entirety without excising that portion of the pulmonary vein lying outside the pericardium. In several instances the entire pulmonary vein has been removed and the site of entrance of the pulmonary vein into the left atrium closed by suturing the wall of the auricle itself. Occasionally histologic examination of the removed lung has shown that either the pulmonary veins or the bronchus had been divided through cancer tissue. With regard to the bronchus, such unfortunate occurrences can be diminished by dividing the bronchus as far as possible from the growth, in some instances even removing a portion of the trachea. As regards the pulmonary veins, it is obvious that routine division within the pericardial sac, in the case of all hilar growths, will diminish the likelihood of leaving behind neoplastic tissue.

Since surgical procedures within the pericardium are of recent origin, very little of the surgical anatomy of the region has been recorded in the literature. It was thought that dissection of the great vessels within the pericardium in a large number of bodies might reveal some anatomic facts of interest to surgeons.

Dissections were carried out on 185 adult cadavers. Seventy of these were fresh autopsy specimens. Estimates were made of the proportion of the circumference of the pulmonary vessels which lay within the pericardium. That part of the circumference of the great vessels covered by serous pericardium was estimated in tenths of the total circumference. The point of bifurcation of the pulmonary artery, the presence of anomalous pulmonary veins, and the presence or absence of a patent vessel in the fold of Marshall were also observed and

From the Department of Surgical Research of the Jefferson Medical College.
Received for publication Nov. 18, 1949.

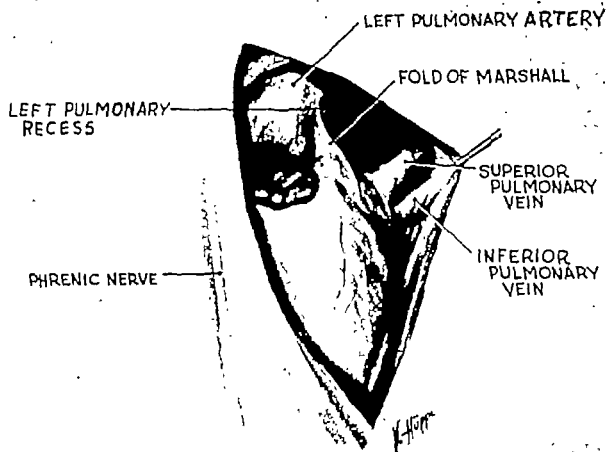


Fig. 3.—The left pulmonary recess.

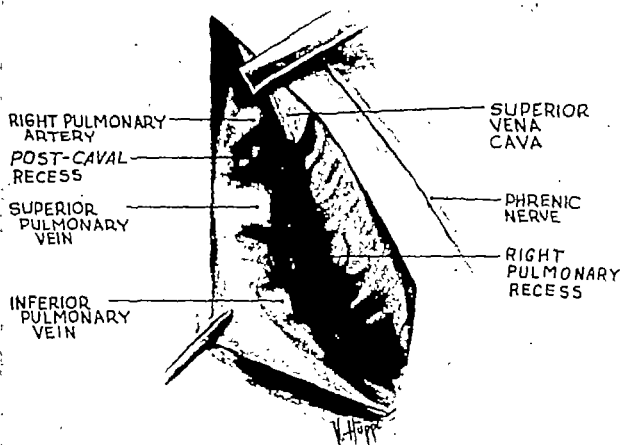


Fig. 4.—Right side of the pericardial sac revealing the postcaval and right pulmonary recesses.

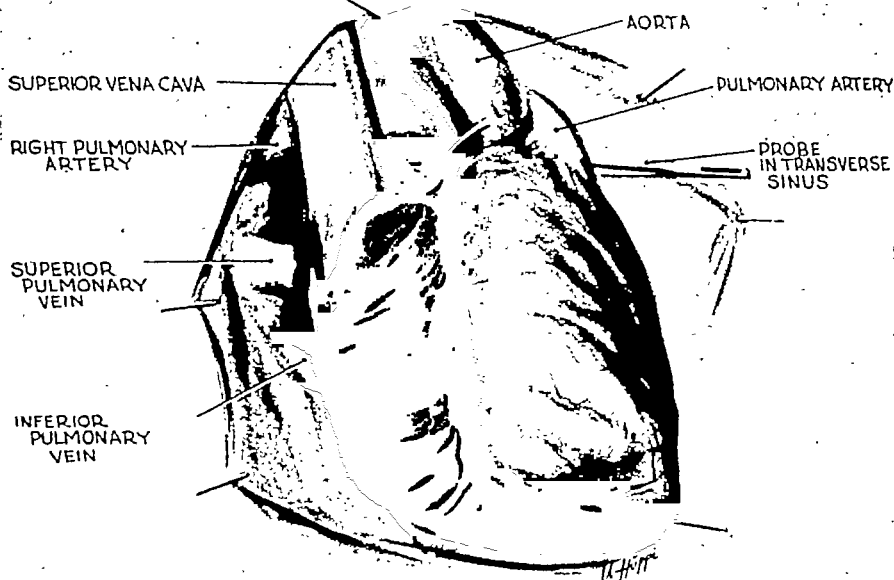


Fig. 1.—Drawing illustrating the transverse sinus and the reflections of the serous pericardium on the right side. A probe is inserted into the transverse sinus pulling the aorta and pulmonary artery to the left.

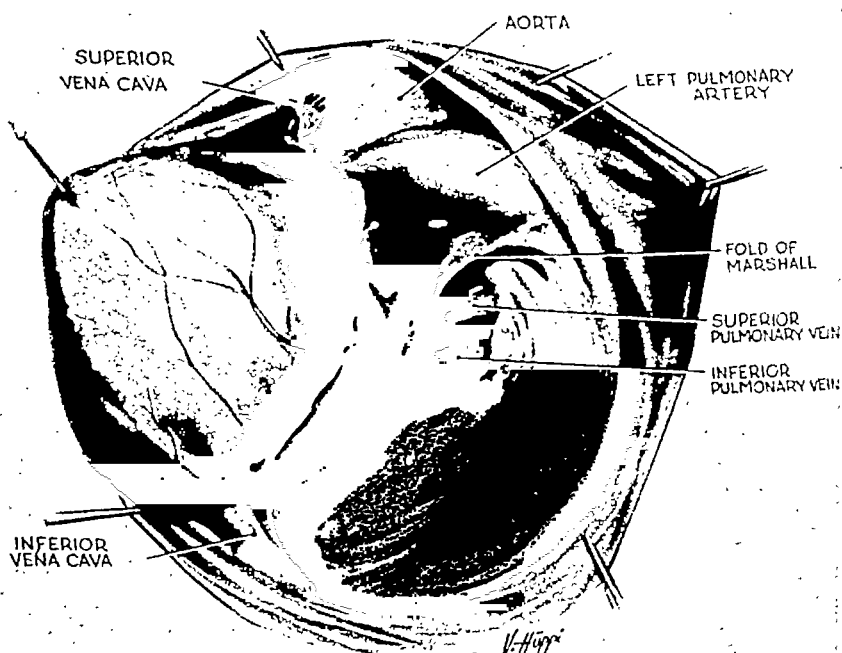


Fig. 2.—The oblique sinus and the serous reflections on the left side of the pericardial sac. Apex of the heart is pulled upward and to the right.

TABLE I. PROTRUSION OF PULMONARY VESSELS INTO THE PERICARDIAL SAC
(Left Side 181 Cases, Right Side 184 Cases)

VESSEL	NUMBER OF CASES WITH VESSELS FOUND IN PERICARDIUM		CIRCUMFERENCE OF VESSEL IN PERICARDIUM	
	LEFT SIDE (PER CENT)	RIGHT SIDE (PER CENT)	LEFT SIDE (PER CENT)	RIGHT SIDE (PER CENT)
Superior pulmonary vein	75	97	67	62
Inferior pulmonary vein	75	46	82	30
Common pulmonary vein	25	3	88	50
Pulmonary artery	100	90* 94†	57	72* 21†

*In transverse sinus medial to superior vena cava.

†In postcaval recess lateral to superior vena cava.

VENOUS ANOMALIES

The reflections upon the left pulmonary veins as described previously occurred in 135 of the 181 bodies observed. In 46 cadavers (25 per cent) the left pulmonary veins merged to form a common trunk which entered the left atrium (Fig. 5). When this single trunk was present on the left side, more than four-fifths of its circumference protruded into the pericardial sac (Table I). On the right side we have seen six common pulmonary veins (3 per cent), and about one-half of the circumference of each of these was visible within the pericardium.

TABLE II. INCIDENCE OF COMMON PULMONARY VEINS

AUTHOR	TOTAL BODIES	LEFT COMMON PULMONARY VEIN		RIGHT COMMON PULMONARY VEIN	
	(NUMBER)	(NUMBER)	(PER CENT)	(NUMBER)	(PER CENT)
Adachi	91	5	5.5	6	6.6
Healey and Gibbon	185	46*	25.4	6†	3.3

*One hundred eighty-one bodies studied.

†One hundred eighty-four bodies studied.

Von Sommerrung²⁷ stated that a common pulmonary vein was more frequently found on the left side. Goeppert¹² in his earlier writings stated that the pulmonary veins joined to form a common trunk more frequently on the right side, but retracted this statement in his later articles. Adachi¹ found 11 common pulmonary veins in 91 cadavers. Table II summarizes our findings and those of Adachi. A left common pulmonary vein was present in about one-fifth of the bodies, while a right common pulmonary vein was present in less than 5 per cent. Aside from Adachi's report, no other statistical study could be found in the literature.

TABLE III. SITE OF FORMATION OF COMMON PULMONARY VEINS

	HEALEY AND GIBBON		ADACHI	
	(NUMBER)	(PER CENT)	(NUMBER)	(PER CENT)
Intrapericardial	4	8	0	0
At edge of sac	40	77	6	55
Extrapericardial	8	15	5	46

On the right side, beginning cephalad on the aorta, the pericardium is reflected in an oblique manner across the superior vena cava descending caudad from medial to lateral (Fig. 1). It then passes to the lateral and posterior surfaces of the superior vena cava and comes to lie upon the right pulmonary artery in the postcaval recess. Only a portion of the anterior surface, or about one-fifth the circumference of the latter vessel is seen at this point. In eight instances the pericardial reflection upon the superior vena cava occurred so low that access to the postcaval recess (Fig. 4) was impossible. In these cases the right pulmonary artery was excluded from this recess, but was readily accessible in the transverse sinus, where it runs transversely posterior to the aorta and superior vena cava (Fig. 1). In fact, we have been impressed with the ease with which the right pulmonary artery may be exposed in this location. Here three-quarters of the circumference of the right pulmonary artery projects into the pericardial sac as compared with the projection of about one-fifth of the circumference in the postcaval recess (Table I).

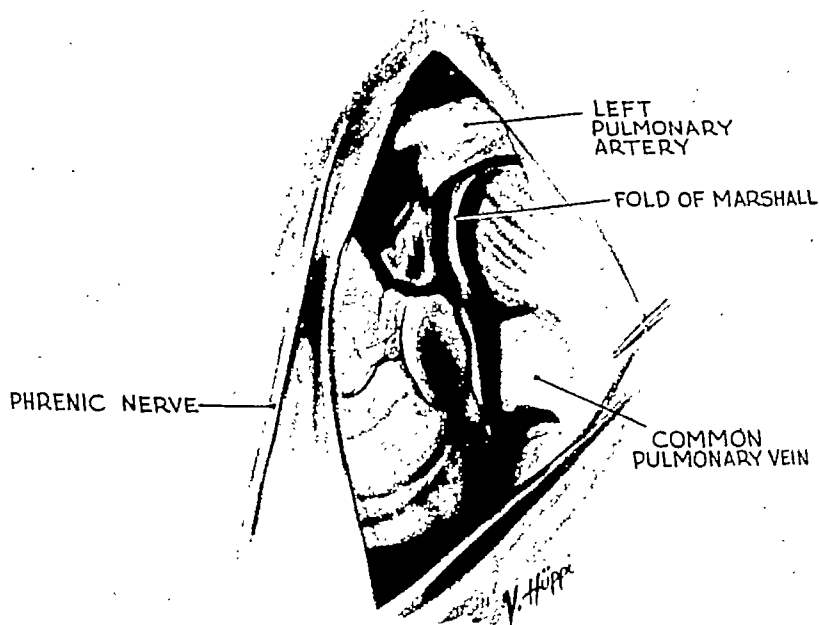


Fig. 5.—Left common pulmonary vein.

The serous pericardium then descends in the recess and reflects upon the superior, anterior, and inferior surfaces of the right superior pulmonary vein, of which about two-thirds projects into the sac. Continuing caudalward in the right pulmonary recess, the pericardium then covers the inferior pulmonary vein in part. In approximately one-half of the specimens this vessel could not be seen within the pericardial sac. When it was visible only about one-third of the circumference could be seen. The serous layer then descends to the diaphragm.

of the left superior vena cava. This ligament, along with the highest intercostal vein on the left side, and the oblique vein on the left atrium, are the remnants of the embryologic left superior vena cava.¹⁸ These changes come about as illustrated in Fig. 7. The right and left anterior cardinal veins communicate about the eighth week, and the venous blood is thereby shunted to the right side. This oblique channel becomes the left innominate vein. The remainder of the left anterior cardinal vein may then be divided as to its future development into three portions; the most cephalic portion becomes the highest intercostal; the medial portion, the ligament of the left superior vena cava; the caudal portion, the oblique vein of Marshall. Conditions may be such, however, that the more primitive structures remain as a left superior vena cava.

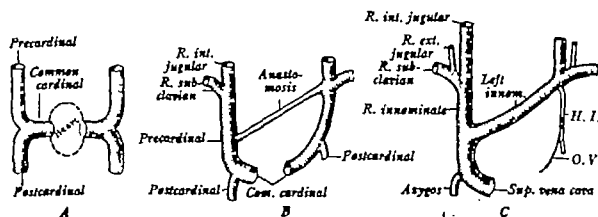


Fig. 7.—Illustration showing the transformation of the cardinal veins of human embryos. A, at six weeks; B, at eight weeks; C, adult. H.I., highest intercostal; O.V., oblique vein of the left atrium. (Reproduced from Arey's "Developmental Anatomy," Philadelphia, 1946, W. B. Saunders Co., with permission of the author and publisher.)

This vessel may be present with or without the presence of the right superior vena cava. The latter is rare and is termed "situs inversus of the superior vena cava." Halpert¹⁴ has reviewed these cases and found only 23 in the literature up to 1942. Since that time 2 additional cases have been reported.^{4, 28} When the right superior vena cava is present, the left is usually of small size. Chouke,⁹ in 1939, in reviewing the literature, found a total of 205 cases of persistent left superior vena cava. He failed to mention, however, the 4 cases cited by Adachi¹ in 1933, and the case reported by Mergoni²⁰ in 1938. We have found reference to 12⁹, 10, 11, 13, 16, 17, 19, 22, 24, 25, 26 in the literature from 1939 to date, and have found 3 in our dissections, bringing the total number to 225 cases of persistent left superior vena cava. Each of the persistent left superior vena cavae in our series were very small in caliber. They were all patent throughout as demonstrated by injection of dye. All were continuous below with the coronary sinus. Two communicated above with the highest intercostal vein and one communicated directly with the left innominate vein (Fig. 8).

We have searched for a patent vessel in the fold of Marshall in 91 cadavers and have found twelve. Three of these, described previously, were patent throughout. The remaining 9 were continuous below with the oblique vein of the left atrium, but extended cephalad only to the superior extension of the vestigial fold. These anomalies must be borne in mind when proceeding to divide the fold as Allison suggests.

These common pulmonary veins may be formed outside of the pericardium, at the edge of the pericardial sac, or within the pericardium. Table III lists the site of junction of the superior and inferior veins to form a common trunk in our 52 cases and in Adachi's 11 cases. In the great majority of cases, the common pulmonary vein was formed at the edge of the pericardial sac.

Another venous anomaly was encountered in 3 of the 185 dissections. In these bodies three separate vessels entered the left atrium, one from each lobe of the right lung. Brantigan⁵ described 2 such cases which he observed at operation.

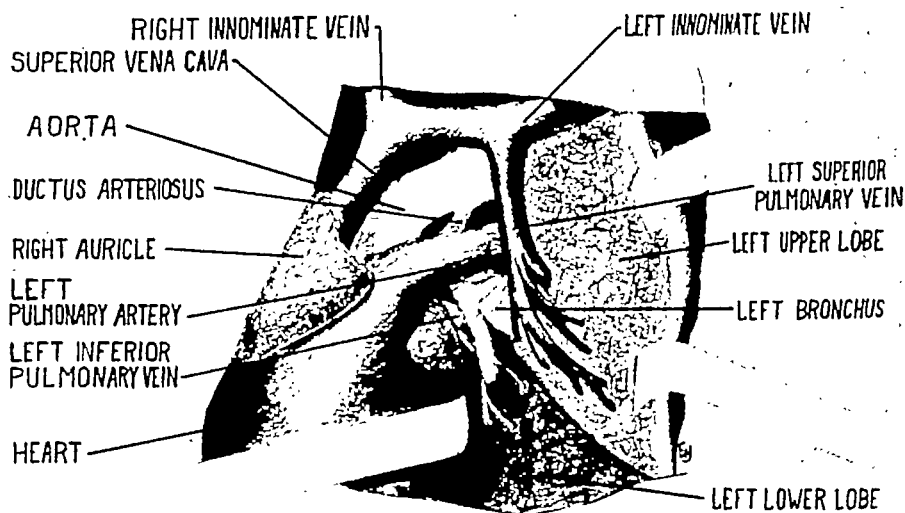


Fig. 6.—Anomalous vessel found in our dissections. Left superior pulmonary vein drains into the left innominate. The left inferior pulmonary vein follows its normal path of drainage into the left atrium.

In one of our dissections we encountered an interesting anomalous connection between the pulmonary and systemic venous system (Fig. 6). This consisted of a large vein draining the entire upper lobe of the left lung and emptying into the left innominate vein. The inferior pulmonary vein entered the left atrium in its usual manner. Brody⁶ in a review of the literature found 68 cases in which there was a partial drainage of pulmonary venous blood into the systemic venous system. Eight of the 68 cases were similar to the one just described. Since that time one other case has been reported by Hughes and Rumore.¹⁵ The embryologic basis of these anomalous communications between the pulmonary and systemic systems and the variations in the number of pulmonary veins entering the left side of the heart has been adequately discussed by Brown,⁷ Patten,²³ and Brantigan.⁵

THE FOLD OF MARSHALL

Allison² stated that "one may further free the upper (left pulmonary) vein by dividing the fold of Marshall." This fold is located between the pulmonary artery and the left superior pulmonary vein (Fig. 2) and contains the ligament

It should be borne in mind that, on the left side, the superior and inferior pulmonary veins join to form a common trunk within the pericardial sac in about one-fifth of all individuals. This simple and common anatomic variation has not been sufficiently emphasized in the past. The presence of anomalous connections between the pulmonary and systemic veins are relatively rare, but the possibility of such connections should not be forgotten. The three persistent left superior venae cavae encountered in our dissections were all small, though patent, and would not have proved troublesome to the operating surgeon.

SUMMARY

1. The intrapericardial anatomy in 185 adult cadavers has been studied. The usual anatomic findings and the common variants have been tabulated and illustrated.

2. It is suggested that, in performing pneumonectomy for cancer of the lung, except in the early cases or the peripheral growths, intrapericardial ligation and division of the pulmonary vessels will provide a wider margin of safety beyond tissues grossly involved by the tumor.

REFERENCES

1. Adachi, B.: *Anatomie der Japaner: II. Das Venensystem der Japaner*, Verlag der Kaiserlich-Japanischen Universität, Kyoto, 1933.
2. Allison, P. R.: Intrapericardial Approach to the Lung Root in the Treatment of Bronchial Carcinoma by Dissection Pneumonectomy, *J. THORACIC SURG.* 15: 89, 1946.
3. Arey, L. B.: *Developmental Anatomy*, Philadelphia, 1946, W. B. Saunders Company.
4. Bestard, A.: Absence of Right Superior Vena Cava and the Presence of a Left Superior Vena Cava, *An. Pnc. de cien. med. Asuncion* 5: 397, 1945.
5. Brantigan, O. T.: Anomalies of the Pulmonary Veins; Their Surgical Significance, *Surg., Gynec. & Obst.* 84: 653, 1947.
6. Brody, H.: Drainage of the Pulmonary Veins Into the Right Side of the Heart, *Arch. Path.* 33: 221, 1942.
7. Brown, A. J.: The Development of the Pulmonary Vein in the Domestic Cat, *Anat. Rec.* 7: 299, 1913.
8. Castellanos, A., and Garcia-Lopez, A.: Case With Four Embryonic Venae Cavae, Two Superior and Two Inferior Cavae, *Bol. Soc. cubana de pediat.* 16: 463, 1944.
9. Chouke, K. S.: Case of Bilateral Superior Vena Cava in the Adult, *Anat. Rec.* 74: 151, 1939.
10. Dohn, B.: Persistent Left Superior Vena Cava, With Report of Case, *Acta path. et microbiol. Scand.* 21: 411, 1944.
11. Fernandez, D., Munist, L., and Gonzalez, J.: Tetralogy of Fallot in Complete Heterotaxia Associated With Other Anomalies, *Arch. Soc. argent. de anat. norm. y Pat.* 7: 233, 1945.
12. Goeppert, H.: *Anatomie des Menschen*, Gegenbauer 7te. Auflage Bd. 2: 3-203, 1899.
13. Goltman, D. W., and Stern, N. S.: Congenital Heart Disease; Report of a Case of Dextroposition, Persistence of an Early Stage of Embryonic Development of the Heart, etc., *Am. Heart J.* 18: 176, 1939.
14. Halpert, B.: Situs Inversus of the Superior Vena Cava, *Arch. Path.* 33: 75, 1942.
15. Hughes, C., and Rumore, P.: Anomalous Pulmonary Veins, *Arch. Path.* 37: 364, 1944.
16. Huseby, R. A., and Boyden, E. A.: Absence of Hepatic Portion of Inferior Vena Cava With Bilateral Retention of Supracardinal System, *Anat. Rec.* 81: 537, 1941.
17. Kernan, J. D.: Obstruction of Right Bronchus Due to Congenital Maldevelopment of the Veins, *Ann. Otol. Rhin. & Laryng.* 53: 817, 1944.
18. Marshall, J.: On Development of the Great Anterior Veins in Man, etc., *Phil. Tr., Lond.* 140: 133, 1850.
19. McManus, J. F. A.: Case in Which Both Pulmonary Veins Emptied Into Persistent Left Superior Vena Cava, *Canad. M. A. J.* 45: 261, 1941.

An interesting relationship exists between the vestigial fold and the bifurcation of the pulmonary artery. Of the 181 bodies observed, the bifurcation occurred at the point of attachment of the fold to the pulmonary artery in 98. The artery bifurcated medial to the attachment in 56 cases, and on the lateral side of the fold in 14 cases. No fold could be found in 13 bodies.

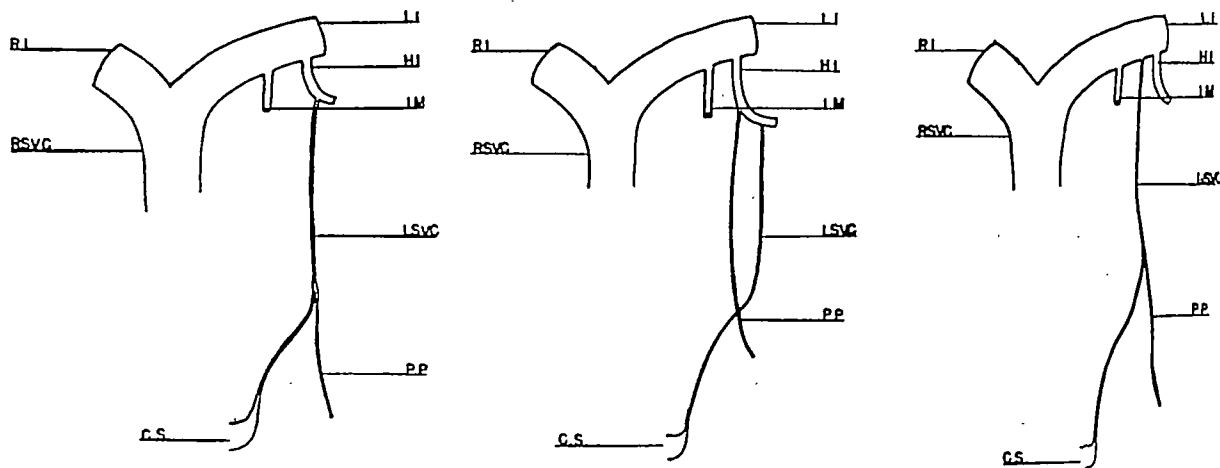


Fig. 8.—Three cases of persistent left superior vena cava observed in our dissections. *RI*, right innominate; *LI*, left innominate; *RSVC*, right superior vena cava; *HI*, highest intercostal; *IM*, internal mammary; *LSVC*, left superior vena cava; *PP*, pericardio-phrenic; *CS*, coronary sinus.

DISCUSSION

With regard to the sinuses and recesses of the pericardium, it should be borne in mind that the right pulmonary artery is readily accessible in the transverse sinus between the superior vena cava and the aorta. Approximately three-fourths of the circumference of the vessel lies within the pericardial sac in this location. Where there is a large growth in the right main bronchus or the right upper lobe it may be difficult to expose a sufficient length of the pulmonary artery lateral to the superior vena cava to ligate and divide the vessel safely. In such cases the right pulmonary artery can be easily exposed by retracting the vena cava laterally and ligating and dividing the vessel between the vena cava and the aorta. In a patient, operated upon by one of our associates (F. F. Allbritten, Jr.), exposure and ligation of the artery in this location proved to be a lifesaving measure. Exposure was difficult because of a large tumor mass and hemorrhage occurred from the stump of the pulmonary artery, which had been ligated and divided lateral to the superior vena cava. The hemorrhage was controlled by digital pressure. The proximal portion of the vessel was then easily exposed by retracting the vena cava laterally, and the artery was safely ligated in this position.

The extent to which the pulmonary veins are covered by the serous layer of the parietal pericardium (Table I) is of minor surgical significance, as in order to pass a ligature around any of the pulmonary veins within the pericardium, the pericardial sac must be penetrated both above and below the vessel being ligated.

MUCOCELLULAR PAPILLARY ADENOCARCINOMA OF THE LUNG

LOBECTOMY, FIVE-YEAR FOLLOW-UP

KERMIT E. OSSERMAN, M.D., AND HAROLD NEUHOF, M.D.
NEW YORK, N. Y.

IN AUGUST, 1946, we reported a case of "Mucocellular Papillary Adenocarcinoma of the Lung," treated by lobectomy. The purpose of the report was twofold: first, to set forth the distinctive microscopic features of the lesion, and second, the multilocular character and pronounced circumscription of the tumor, occupying most of the lower lobe.



Fig. 1.—Nov. 21, 1949, film five years after operation. Elevated diaphragm due to phrenic nerve severance at operation.

To summarize the case, the patient was 44 years old, female, first seen by us in 1944. There was a history of cough starting in 1941, which became productive in 1943. Physical examination showed that the lungs were clear except beneath the left scapula where an area of dullness was detected at the level of the fifth thoracic vertebra. There was clubbing of the fingers. Roentgenogram of the chest showed a homogeneous density in the left lower lobe, circumscribed and lobulated. An exploratory thoracotomy was performed in November, 1944. At operation, the left lower lobe was found to be greatly expanded by a circumscribed lobulated tumor. There was no cancerous involvement of the pleura or any of the hilar lymph nodes. A dissection lobectomy of the left lower lobe was performed. The pathology report was mucocellular papillary adenocarcinoma of the lung, histologically of the type of the multicentric papillary adenocarcinoma. The report of this case was made two years after operation, at which time the patient was symptom free.

In a paper entitled, "Alveolar Cell Carcinoma of the Lung (Pulmonary Adenomatosis, Jagziekte)," Delarue and Graham² stated in reference to our

20. Mergoni, G.: Case of Persistent Left Superior Vena Cava, *Cuore e circolaz.* 22: 604, 1938.
21. Morris, Sir Henry: Human Anatomy, ed. 10, Philadelphia, 1945, The Blakiston Company.
22. Nishii, H., and Suga, Y.: Double Superior Vena Cava: Autopsy Findings, *Okayama-Igakkaie-Zasshi* 55: 1421, 1939.
23. Patten, B. M.: Human Embryology, Philadelphia, 1946, The Blakiston Company.
24. Prows, M. S.: Two Cases of Bilateral Superior Venae Cavae, One Draining Closed Coronary Sinus, *Anat. Rec.* 87: 99, 1943.
25. Sanders, J. M.: Bilateral Superior Vena Cava, *Anat. Rec.* 94: 647, 1946.
26. De Sigueira, M. E., and Zaragoza, A.: Two Venous Variations: Single Left Superior Vena Cava and Persistence of Left Superior Vena Cava, *An. Fac. de med. da Univ. de São Paulo* 19: 27, 1943.
27. Von Sommerrung, W.: *Angiologie*, 5: 334, 1841.

THORACOPLASTY AND STREPTOMYCIN IN THE TREATMENT OF TUBERCULOUS EMPYEMA

MAJOR MAURICE R. CONNOLLY, AND COLONEL JAMES H. FORSEE
MEDICAL CORPS, UNITED STATES ARMY

THE treatment of tuberculous empyema has long been characterized by poor results. Since streptomycin became available in adequate quantities at Fitzsimons General Hospital late in 1946, a distinct evolution in the surgical management of pulmonary tuberculosis at this hospital has developed. The time interval from the onset of the disease to the application of thoracoplasty has been appreciably shortened. Many patients with cavernous caseopneumonic disease have responded to streptomycin therapy with a marked resolution of the exudative element making thoracoplasty feasible at a relatively early period in the disease process.¹ A definite improvement has also been reflected in the results obtained in the treatment of tuberculous empyema.

This report pertains to a consecutive group of 22 patients having tuberculous empyema treated by extrapleural thoracoplasty and streptomycin during the period Dec. 1, 1946, to Aug. 1, 1948. In all instances the pleural fluid was purulent and on culture was positive for acid-fast bacilli in 20 of the 22 patients. The following classification of tuberculous empyema is used in this hospital and is as follows:

1. Tuberculous empyema with no evident parenchymal disease.
2. Mixed (that is, pyogenic plus acid-fast bacilli) tuberculous empyema with no evident parenchymal disease.
3. Tuberculous empyema with parenchymal disease.
4. Mixed tuberculous empyema with parenchymal disease.

In our series, 7 of the patients are in Group 3 and 15 in Group 4. No patients were in Group 1 or 2. All patients in Group 4 were seriously ill. As experience increased in the use of streptomycin the time interval between the onset of the empyema and the application of thoracoplasty in conjunction with streptomycin was gradually decreased. Ten of the surviving patients in Group 4 have had arrest of their parenchymal disease and obliteration of the empyema space. An illustrated case report among Group 4 patients includes the following:

CASE 1.—A 24-year-old male soldier gave a history of pneumonia in 1942, from which he recovered without complications. There were no other serious illnesses and no history of contact with tuberculosis until 1947. While stationed in Germany in January, 1948, he began to complain of undue fatigue, productive cough, blood-tinged sputum, and chest pain. He was immediately hospitalized. On the day following admission he had a sudden onset of severe dyspnea, hemoptyses, chills and fever, and marked prostration. Roentgenograms confirmed the presence of a spontaneous pneumothorax with approximately 50 per cent collapse of the lung with large cavitation in the left upper lobe. Daily thoracentesis with the removal of air and fluid was accomplished with moderate relief of symptoms. Closed tube

¹From the Surgical Service, Fitzsimons General Hospital, United States Army, Denver, Colo.

Received for publication Jan. 5, 1950.

case, "The result seems to indicate that a two-year period of freedom from manifestation of symptoms after lobectomy gives no assurance of complete eradication of the disease." It is, therefore, of interest to report a follow-up on our patient, five years after operation in November, 1944. The patient has been seen at intervals during these five years. In November, 1949, the patient, 49 years of age, was symptom free. Physical examination showed weight to have remained stationary (128 pounds). Hemoglobin was 14.6 Gm. No clubbing of fingers. No adenopathy. No excursion of the left leaf of the diaphragm, as evidenced by percussion and fluoroscopy (phrenic nerve severance at operation). The lungs were clear and resonant. Roentgenogram of the chest taken Nov. 21, 1949, was negative (Fig. 1). The branches of the pulmonary artery were seen to be spread somewhat to follow the expanded upper lobe.

REFERENCES

1. Osserman, K. E., and Neuhof, H.: J. THORACIC SURG. 15: 272, 1946.
2. Delarue, N., and Graham, E. A.: J. THORACIC SURG. 18: 237, 1949.

There have been four deaths among the 15 patients of the Group 4 classification and no deaths among the Group 3 patients. The over-all fatality rate for the 22 patients with tuberculous empyema and parenchymal disease was 18 per cent. This compares most favorably with the data reported from this hospital by Leaver and Hardaway in 1937, as they reported a mortality rate of 70.9 per cent in 24 patients suffering from mixed tuberculous empyema and 22.9 per cent in 35 patients having a tuberculous empyema.² The deaths in our series presented conditions which to date we have been unable successfully to overcome. One patient, a 21-year-old soldier, developed a postpneumonic empyema while a prisoner of war in Germany. Several operative procedures including an extensive decortication and unroofing of the empyema cavity were accomplished. The diagnosis of tuberculosis was made by the examination of the surgical specimen of diseased pleura. Streptomycin was first given two years after the onset of the illness. He died three years after his first operation performed while a prisoner of war in 1944. Post-mortem examination revealed widespread tuberculous involvement of the viscera. A second death occurred in a patient who was a severe drug addict and had been chronically ill for twelve years with pulmonary tuberculosis. He died on the fourth postoperative day following a first-stage thoracoplasty. The third patient died approximately one year after obliteration of the empyema cavity by thoracoplasty, from progressive contralateral tuberculous disease with giant cavitation. Post-mortem examination revealed that the empyema space had been obliterated. The fourth death occurred in a 51-year-old man who had had pulmonary tuberculosis treated by artificial pneumothorax for 22 years. He had a first-stage thoracoplasty performed six months after the onset of a mixed infection empyema and died from a coronary thrombosis on the eleventh postoperative day.

Ten patients with mixed tuberculous empyema and parenchymal disease have arrested their disease and have had no reactivation during an average period of 24.5 months following thoracoplasty with streptomycin therapy. The shortest follow-up period was 14 months, the longest 32 months. One patient had active tuberculous disease in the contralateral lung 18 months following thoracoplasty. The empyema space had been obliterated.

Seven patients were in Group 3, that is, pure tuberculous empyema with parenchymal disease. Six patients have had arrest of their parenchymal disease and no evidence of empyema since operation. The average length of the follow-up period has been 18.4 months. The shortest period was 13 months, the longest 29 months. The following case report of this group which has not had arrest of the disease process is recorded.

CASE 2.—A 32-year-old white male soldier contracted malaria, in 1942, on Bataan and was seriously ill. He was later captured by the Japanese and while a prisoner of war in the Philippine Islands he developed beriberi and lost approximately 40 per cent of his normal weight during imprisonment. He was liberated on Aug. 20, 1945. In March, 1947, he was hospitalized complaining of symptoms of nasal sinusitis, sensation of fullness in the right ear, a moderate cough productive of one teaspoonful of whitish sputum, weight loss, and generalized fatigue. Roentgenograms of the chest revealed a massive pleural effusion in the left

intercostal drainage was established in January, 1948, and discontinued while enroute to the United States in February, 1948. On Feb. 27, 1948, he was admitted to Fitzsimons. Roentgenograms of the chest revealed a fluid level in the left pleural space, large cavitation on the left, considerable collapse of the left lung, and slight shift of the mediastinum to the right (Fig. 1). Thoracentesis was carried out every other day with the removal of 500 to 1,000 c.c. of thick greenish fluid having a specific gravity of 1.021 which was positive on culture for acid-fast bacilli. One-half gram of streptomycin was placed in the pleural space each time fluid was removed. The patient improved symptomatically with a sustained decrease in fever, better appetite, and a lessening in the accumulation of fluid. Penicillin therapy, 300,000 units in oil daily, was started two days prior to and continued for six days after



Fig. 1.



Fig. 2.

Fig. 1 (Case 1).—Spontaneous pneumothorax, cavitation, and mixed infection, tuberculous empyema, left.

Fig. 2 (Case 1).—Postthoracoplasty. No cavity. No empyema.

each stage. A first-stage posterolateral thoracoplasty was performed April 27, 1948. Ten days later an anterior-stage thoracoplasty was performed. Further staged posterolateral thoracoplasties were accomplished until a ten-rib thoracoplasty had been completed May 18, 1948, with removal of all of the first five ribs and long segments of the sixth to tenth ribs, inclusive. The pleural space was kept dry by frequent thoracentesis between operative stages. Streptomycin intramuscularly, 1 Gm. daily, had been started March 29, and was continued for eighty-seven days. The exudative element of the pulmonary disease showed considerable resolution and by May, 1948, there was no evidence of pleural fluid. Eighteen months after the completion of thoracoplasty the patient was asymptomatic, afebrile, and had no cough or sputum production. Numerous gastric washings have been negative for acid-fast bacilli. X-ray examination of the chest revealed no evidence of parenchymal cavitation, the pleural space was obliterated, and the patient was considered in the arrested phase in reference to tuberculosis (Fig. 2).

pneumothorax, 1 in association with a tuberculous pneumonia, and 2 were of gradual onset without any demonstrable exciting cause. Eight of the series were associated with therapeutic efforts, 7 patients were under treatment by artificial pneumothorax therapy, and 1 followed extrapleural thoracoplasty for parenchymal disease before the availability of streptomycin. During the period of this report 173 patients received 509 stages of extrapleural thoracoplasty, 63 patients received closed intrapleural pneumolysis, and 25 patients had lobectomy, partial or complete, or pneumonectomy performed at Fitzsimons in the treatment of pulmonary tuberculosis.

Therapy in relation to the type of onset and the presenting pathology of the empyema is significant. Patients with a sudden onset, a spontaneous pneumothorax, bronchopleural fistula, and rapid reaccumulation of fluid or extreme sepsis may require open or closed drainage as a lifesaving measure. Seven of our patients belonged in this group. They were all in Group 4 and were critically ill. In 8, the onset of the empyema was sudden but did not require open or closed drainage. The accumulation of empyema fluid in these patients was kept under control with frequent aspirations so that thoracoplasty could be completed without recourse to drainage. It was in this group that streptomycin and early thoracoplasty markedly lessened the morbidity. Seven had a mixed infection empyema and 1 had a pure tuberculous empyema. The patients who had a gradual onset of the empyema did not receive drainage before thoracoplasty.

We have had only limited experience with decortication for the treatment of tuberculous empyema. The encouraging results reported by Weinberg and Davis³ offer further hope for improving the results in the treatment of this condition.

SUMMARY

Tuberculous empyema is still a dreaded complication of pulmonary tuberculosis. The employment of streptomycin in conjunction with extrapleural thoracoplasty has appreciably decreased the mortality rate in tuberculous empyema. The general trend in the earlier application of extrapleural thoracoplasty and the judicious use of excisional surgery along with streptomycin in the management of pulmonary tuberculosis constitutes, we believe, major advances in the therapy of this disease. The results in the treatment of 22 consecutive patients with tuberculous empyema with thoracoplasty and streptomycin is very encouraging. The mortality rate was 18 per cent. Seventeen are alive and their disease arrested. One patient still has active parenchymal disease. The postoperative follow-up period in the entire group varies from 13 to 32 months.

REFERENCES

1. Forsee, James H.: Extrapleural Thoracoplasty Early in Caseo-pneumonic Tuberculosis, *Rocky Mountain M. J.*, June, 1949.
2. Leaver, F. Y., and Hardaway, R. M.: Empyema Complicating Artificial Pneumothorax, *Am. Rev. Tuberc.* 35: 538, 1937.
3. Weinberg, Joseph, and Davis, J. Dwight: Pleural Decortication in Pulmonary Tuberculosis, *Am. Rev. Tuberc.* 60: 288, 1949.

hemithorax and a large 5 by 8 cm. cavity in the left upper lung field (Fig. 3). He was transferred to Fitzsimons General Hospital April 10, 1947. Sputa were positive on culture for acid-fast bacilli. Thoracenteses twice weekly resulted in gradual diminution in the amount of pleural fluid. He received streptomycin 2 Gm. daily from June 14, to Aug. 6, 1947, when it was discontinued due to marked dizziness. He was restarted in the dosage of 1 Gm. per day on August 14, and continued until Nov. 3, 1947. By the end of the first course of streptomycin the patient's symptoms had markedly decreased (Fig. 4). Because of the persistent empyema and cavitation, thoracoplasty was performed with removal of seven ribs in three stages at intervals of 21 days. The last stage was performed on April 8, 1948. Penicillin was given as in the first case. A total of 294 Gm. of streptomycin was given. A follow-up report, eighteen months postoperatively from a Veterans Hospital indicated that sputum was positive but no empyema space was demonstrable. Lobectomy of the left upper lobe may be advisable.



Fig. 3.

Fig. 3 (Case 2).—Giant parenchymal cavity, empyema, left.

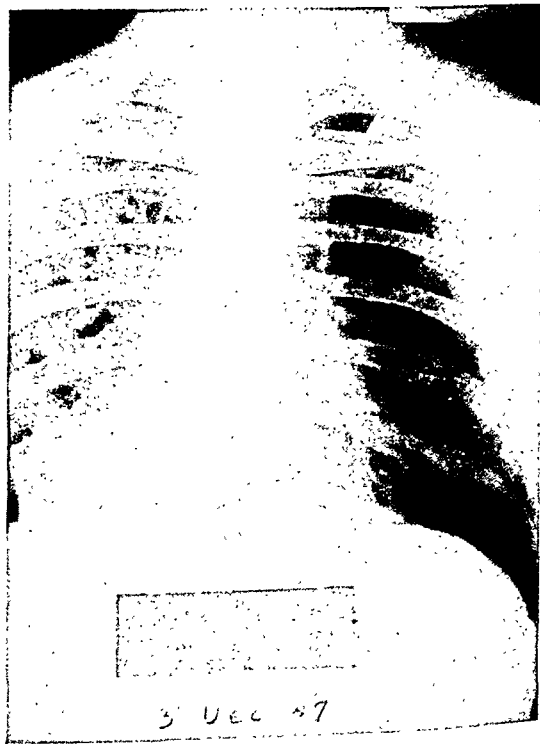


Fig. 4.

Fig. 4 (Case 2).—Prethoracoplasty. Considerable clearing of parenchymal lesions and of empyema.

The changing concepts of therapy and improvements in the management of pulmonary tuberculosis are, we believe, partially reflected in analyzing the precipitating causes of tuberculous empyema noted in this series of patients. The earlier abandonment of an unsuccessful pneumothorax, the earlier application of thoracoplasty in streptomycin treated patients, and the wider acceptance of excisional surgery in this disease are encouraging. In our series of 22 patients, 14 developed a tuberculous empyema as a direct accompaniment of their disease unrelated to any therapeutic efforts. Of this group, 3 developed from a massive pleural effusion, 8 following a spontaneous

On Sept. 7, 1948, the patient was delivered spontaneously without anesthesia, of a live, male child, weighing 6 pounds, 12 ounces. The total duration of labor was fourteen hours, thirty minutes, the placenta and membranes were expelled completely and spontaneously. The condition of mother and child after delivery was excellent. The puerperium was normal and uneventful.

One year, approximately, following delivery, mother and child reported for examination. Roentgenograms taken of the mother's chest showed no deterioration of the lung lesion. The intradermal tuberculin sensitivity test (1:1,000) in the child was negative.

CASE 2.—Mrs. J. S., aged 28 years. This patient, approximately five months pregnant, was admitted to Robroyston Hospital, Glasgow, on July 7, 1948. Several weeks prior to admission she complained of slight pain, localized to the left posterior chest wall. Morning cough accompanied by scanty sputum, mucoid in character, was also present.

In 1947, she received sanatorium treatment for an active, left-sided lung lesion, namely, had rest supplemented by a left side phrenic nerve interruption. Attempts at artificial pneumothorax on this same side had been tried without success.

On the patient's admission to Robroyston Hospital the lung lesion, clinically and radiologically, was found to be fairly fibrotic, but a cavity was present over the left apex and she had a positive sputum. Sedimentation rate was 18 mm. at the end of one hour (Westergren). Clinical examination of other vital organs failed to reveal any abnormalities. Pelvic examinations confirmed the pregnancy but did not disclose any obvious cause for an abortion in 1946.

The first stage of a left-sided, seven-rib thoracoplasty was performed on July 12, 1948, the second being completed fourteen days later. No complications were encountered during both operative procedures, and recovery was uneventful.

On Nov. 19, 1948, this patient had a spontaneous delivery at full term of a live, male child, after a labor which lasted ten hours, thirty minutes. The placenta and membranes were expelled completely, spontaneously, ten minutes later. The birth weight of the child was 5 pounds, 3 ounces.

Examination of the mother and child ten months after the confinement was satisfactory. Roentgenograms of the mother's chest showed no progression of the disease, sedimentation rate was 4 mm. (Westergren), and sputum remained negative for the tubercle bacillus. The intradermal tuberculin sensitivity test (1:1,000) in the child was negative.

CASE 3.—Mrs. S. McC., aged 28 years. This patient, five months pregnant, and complaining of a slight unproductive cough, was admitted to Robroyston Hospital, Glasgow, on Feb. 13, 1949. The lung lesion, confined to the right upper zone, was fairly fibrotic in type with cavity formation, and sputum was positive for the tubercle bacilli. Sedimentation rate was 14 mm. at the end of one hour (Westergren).

In 1943, she received routine sanatorium treatment for ten months, including right-sided artificial pneumothorax and phrenic nerve interruption to this same side. During the years 1943 to 1949, following discharge, she was remarkably well. On admission to Robroyston Hospital, Glasgow, she was in good general state. Clinical investigation did not reveal any abnormality of the other vital organs. Pelvic examination confirmed the presence of a five months' pregnancy and a contracted pelvis, the presumed cause of a protracted labor with death of the child in 1948. Because of a very great desire on her part to have a live child, and the fact that the lung lesion was dominantly productive in type, unilateral in distribution, general condition being reasonably good, a right-sided thoracoplasty was advised. The first stage was performed on Feb. 14, 1949, the second on Feb. 28, 1949. No complications were met. On May 25, 1949, following artificial rupture of the membrane, patient was delivered, spontaneously, after preliminary episiotomy, of a live female child, weighing 6 pounds, 8 ounces. Duration of labor was three hours, fifty-five minutes. Placenta and membranes were expelled completely, spontaneously, in ten minutes. The puerperium was normal and uneventful.

OBSERVATIONS ON THORACOPLASTY DURING PREGNANCY

J. P. McINTYRE, M.D.

GLASGOW, SCOTLAND

INTRODUCTION

THE revolutionary improvements in medicine in recent years are reflected in the present-day change of outlook on the reciprocal relationship of pregnancy and tuberculosis. Not only has this study gained fresh impetus thereby, but modern concepts of its cognate problems have emerged. By and large, the proprieties of the combined conditions are now generally accepted, the pregnancy assuming a secondary role to that of the associated lung lesion. As a result the value of collapse therapy, both prophylactically for the child, and curatively for the mother, has become considerable. Nevertheless, publications relating to thoracoplasty during pregnancy are singularly few. As yet, only four isolated instances have been reported in international literature (quoting Seeley and associates, 1940). To date, McIntyre (1948) has made the sole contribution to British literature. Accordingly the following examples of thoracoplasty during pregnancy were considered worthy of putting on record in view of their exceptional interest, and the fact that they might contribute to the status of collapse therapy in the pregnant tuberculous patient, as yet, not definitely defined.

ILLUSTRATIVE CASES

CASE 1.—Mrs. M. O'D., aged 38 years. This patient, four and one-half months pregnant, was admitted to Robroyston Hospital, Glasgow, on April 20, 1948, complaining of a productive cough of several weeks' duration.

In 1947, she received sanatorium treatment for a left-sided, active lung lesion. Attempts to establish a left artificial pneumothorax were unsuccessful and consequently a left-sided phrenic nerve interruption was performed. At her own request, she was dismissed from the sanatorium prematurely, after six months' institutional treatment. Her health, for several months following this discharge, was indifferent, cough, tiredness, and night sweats being present.

On admission to Robroyston Hospital her general condition was found to be fairly good. The lung lesion, clinically, was dominantly left sided in distribution, and fibrocaceous in type. This was confirmed by the radiologist who reported no definite disease seen in the right lung. There was marked pleural thickening on the left side. The mediastinum was drawn to the left. Bacteriologic examination of the sputum revealed the presence of many tubercle bacilli. The sedimentation rate was 20 mm. at the end of one hour (Westergren). But for a slight displacement of the heart to the left, clinical examination of other vital organs revealed no gross abnormality. Following induction by sodium pentothal (0.5 Gm.) intravenously, and under general anesthesia maintained by nitrous oxide oxygen and Trilene, administered endotracheally, a two-staged thoracoplasty was performed. The first stage was done on April 23, 1948, the second, May 10, 1948. Seven ribs in all were removed, subperiosteally. No undue bleeding was noticed during these operations, both of which were well tolerated by the patient.

From the Robroyston Hospital.

Received for publication Jan. 13, 1950.

this problem. Of necessity, comments are confined to points of practical interest essential to decisions on the suitability of pregnant phthisical patients for thoracoplasty.

In the first instance valid recommendations for this form of treatment cannot be made without careful consideration of the broad fundamental principles involved. Such are the individual variations of pregnancy and tuberculosis, for example, that attempts to formulate infallible guides to treatment are futile. In effect, an individualistic attitude must be adopted, with factors physical, psychic, economic, and sociologic in nature being most meticulously evaluated. Furthermore, in all eventualities, and because of the issues at stake, the patient, the husband, and if necessary, the relatives must be told frankly and unequivocally of the magnitude of the risks run. Moreover, the important part played by experienced nursing personnel, and the availability of suitable equipment for the effective application of this highly specialized form of treatment are not to be ignored. In addition, and because of the contingencies consequent on the combined conditions, it is desirable that the medical and obstetric supervision of these patients should be the dual responsibility of the consultant in tuberculosis and obstetrics, respectively.

Finally, it should not be necessary to stress the importance of a comprehensive after-care scheme in which help, financially or in kind, is envisaged.

In this present study, estimation of the patient's suitability for this operation, from the clinical aspect, was based on (1) the age of the patient, (2) cardiocirculatory function, (3) period of gestation, with the optimal time for the operation, in the writer's opinion, being the intermenstrual period, (4) the dominantly fibrotic character and extent of the associated lung lesion, (5) the failure of previous collapse therapeutic measures to achieve cavity closure, and finally, (6) the mental make-up of the patient.

Cognizant of the obvious dangers, immediate and remote, associated with the performance of this major operation during pregnancy, and also the fact that obstetric problems may supervene at any time to complicate the issue, the question as to whether thoracoplasty in the pregnant patient is worth while presents itself. It is submitted that in view of the grave potentialities of cavernous lung disease with positive sputum to the community, in general, the patient and offspring, in particular, thoracoplasty during pregnancy, a principle of prevention, no less than cure, might well be further exploited.

SUMMARY

Three instances are recorded in which partial thoracoplasty was performed during pregnancy. All made uneventful recoveries with sputum conversion. Furthermore, pregnancy, parturition, and the puerperium progressed with no apparent difficulty. Despite the reduction in total lung volume, dyspnea was never troublesome. Mother and child were examined at intervals, varying from three months to one year, after delivery. In each instance, both were alive and well.

A follow-up study, approximately three months after delivery, gave gratifying results. Mother and child were both alive and well. No deterioration in the mother's lung lesion was reported by the radiologist. The child weighed 12 pounds, 2 ounces and was obviously thriving. The Mantoux test was negative (1:1,000).

DISCUSSION

The cause and effect relationship of pregnancy and tuberculosis has been a perennial topic for discussion, opinions on the subject varying between wide extremes. Thus, while many maintain that pregnancy influences the course of pulmonary tuberculosis adversely, others believe the converse. In a large series of pregnant and nonpregnant patients, Ornstein and Kovnat (1935) recorded a death rate of 35 per cent among the former, 33 per cent among the latter, the difference in the comparative mortality figures being negligible. This was confirmed by Hill (1928), Barnes and Barnes (1930), Marshall (1931), and Forssner (1924). By and large, the character of the associated lung lesion rather than the pregnancy is now considered the more important factor in prognosis. Moreover, much medical evidence can be adduced to show that with the institution of appropriate and adequate nursing and medical attention, under ideal environmental conditions, early in the antenatal period, pregnant patients with active phthisis need not now fare worse than their nonpregnant sisters. From the results of investigations on twenty-six pregnant patients, treated by artificial pneumothorax, Cutler (1944) concluded that the risks run by the pregnant patient, who is also tuberculous, are commensurate with the quality of the lung collapse obtained from such treatment. Conceding this point, the next logical step in rational therapeutics must surely be the application of other available measures designed to stabilize the diseased process in the lung. In theory, at least, and provided the patient is in good general state, thoracoplasty should be considered for those pregnant patients in whom simpler measures of collapse therapy have previously been tried but have met with no success. Ironically enough, reports on this aspect of the management of the pregnant tuberculous patient are very few. Accordingly, to date, it would seem that the compatibility of thoracoplasty during pregnancy is not generally recognized.

In this present study, not only the practicability, but also the immense prophylactic potentialities of this principle are illustrated. Figures presented, however, are of no importance statistically. On the basis of this investigation, therefore, recommendations for thoracoplasty during pregnancy can only be tentative.

Albeit, the fact that international and national literature, alike, have ignored or lightly dismissed this aspect of the problem as impracticable, results recorded are not to be deprecated. Furthermore, by accepting the author's conclusions that where desirable thoracoplasty during pregnancy may also be technically feasible, a fresh field of speculation on this vexed question of pregnancy and tuberculosis is presented. Unfortunately it is not within the scope of this short monograph to discuss in detail the many variables complicating

AN APPARATUS FOR ANESTHESIA IN EXPERIMENTAL THORACIC SURGERY

C. ROLLINS HANLON, M.D., THOMAS N. P. JOHNS, M.D., AND VIVIEN THOMAS
BALTIMORE, MD.

THE apparatus here described has for some years provided satisfactory anesthesia for thoracic surgery in the Surgical Hunterian Laboratory of the Johns Hopkins Medical School. Numerous requests for details of its construction have occasioned this description.

The apparatus consists essentially of a motor-driven valve which periodically interrupts a stream of air or oxygen passing over an ether reservoir into an endotracheal tube. This provides alternate inflation and deflation of the lungs. An over-all view of the apparatus is seen in Fig. 1.

By means of a rheostat* the motor† may be regulated so that the interrupter valve turns at any rate up to 45 times a minute. A rate of 20 revolutions per minute is usually proper, although higher speeds may be necessary for animals weighing 20 kilograms or more. Compressed air or oxygen enters the valve at 6 (Fig. 1) and emerges through 7 to inflate the lungs. As the valve rotates to allow deflation of the lungs through an exhaust opening, it momentarily blocks the air intake at 6, leading to increased pressure in the system proximal to this point. Compensation for this increased pressure is provided by the expansion of approximately eight feet of medium-walled rubber tubing as well as by compression of the vapor over the ether: if the flow of air is excessive or the tubing nonexpansile, one of the connections may separate. A needle valve at the air source provides control of pulmonary expansion after the thorax has been entered. A safety valve may be interpolated in the air line to prevent overdistention of the lungs, but with ordinary care in adjusting the flow of air this precaution is unnecessary. A diagram of the apparatus is seen in Fig. 2. The detailed sketch of the interrupter valve in Fig. 3 should be self-explanatory to a competent machinist.

The ether reservoir is a Wolfe bottle of one liter capacity. It operates best when about two-thirds full. The glass T tubes extend just through the rubber stoppers which are taped firmly in place. Ether is added through the central opening.

The concentration of ether in the anesthetic mixture may be varied by application of clamps to the tubing surrounding the ether reservoir, as follows (see Figs. 1 and 2):

Clamp at 10	Negligible anesthetic effect
No clamps	Usual maintenance level
Clamp at 8 (Fig. 1)	Fairly heavy ether vapor
Clamps at 8 and 9	Maximal anesthetic effect

With clamps at 10 and 11 there would be no delivery of ether vapor, but a clamp at 11 is inadvisable since it obviates the cushioning effect of the compressible air over the ether. Elimination of such an expansion chamber in the system may lead to respiratory difficulty or to separation of connections proximal to the clamps.

From the Department of Surgery of the Johns Hopkins University and the Johns Hopkins Hospital.

Received for publication May 16, 1949.

*All metal power rheostat, type PR-50 (50 watts, 500 ohms). Made by International Resistance Company, Philadelphia, Pa.

†A speed-reducer motor, type NSE (11 R, 115 volts, A.C. or D.C.). Made by Boline Electric Company, Chicago, Ill.

Acknowledgments.—The author wishes to express his thanks to Dr. M. A. Foulis, Superintendent, Robroyston Hospital, Glasgow, who performed the operations and also to Dr. W. C. Armstrong, consultant obstetrician, Robroyston Hospital, Glasgow, for his expert management of the confinements.

REFERENCES

1. Barnes, H. L., and Barnes, L. R. P.: Pregnancy and Tuberculosis, *Am. J. Obst. & Gynec.* 19: 490, 1930.
2. Cutler, J. W.: The Harmful Influence of Pregnancy on Advanced Tuberculosis as Modified by Collapse Therapy, *Am. J. Obst. & Gynec.* 47: 1, 1944.
3. Forssner, H. J.: Les relations entre l'état de gestation et la tuberculose, *Rev. de la tuberc.* 5: 730, 1924.
4. Hill, A.: Statistical Study of Relation Between Pregnancy and Tuberculosis, *Am. Rev. Tuberc.* 17: 113, 1928.
5. Marshall, G.: Management of Pregnancy, Parturition, Puerperium in Tuberculous Women, *Brit. M. J.* 1: 140, 1931.
6. McIntyre, J. P.: Thoracoplasty in the Tubercle Gravidæ, *Brit. J. Tuberc.* 42: 45, 1948.
7. Ornstein, G. G., and Kovnat, M.: Influence of Pregnancy on Pulmonary Tuberculosis, *Am. Rev. Tuberc.* 31: 224, 1935.
8. Seeley, W. F., Siddall, R. S., and Balzer, W. J.: Pregnancy and Thoracoplasty, *Am. J. Obst. & Gynec.* 39: 51, 1940.

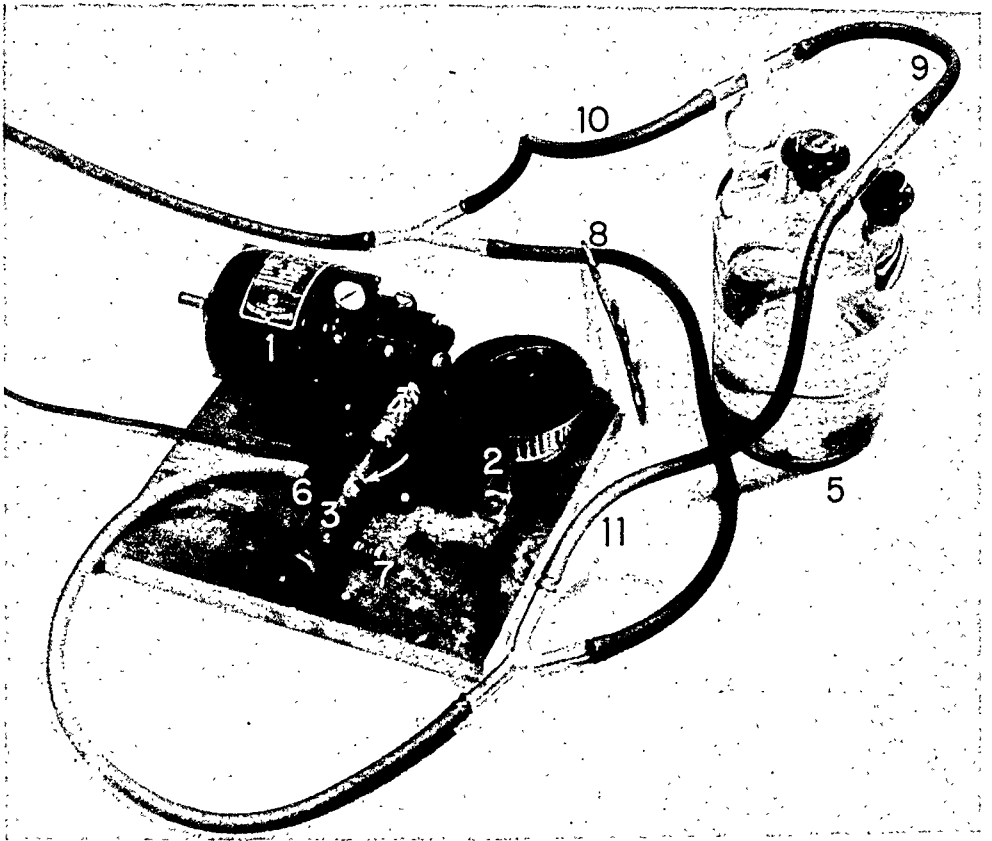


Fig. 1.—Photograph of apparatus showing motor (1), rheostat (2), and interrupter valve (3) connected to motor by means of a flexible coupling (4). Air or oxygen is blown over ether in the Wolfe bottle (5) to enter the valve at (6) and emerge from (7) to enter the tubing leading to the endotracheal catheter. Delivery of ether vapor may be regulated by clamps (8), (9), and (10). During expiration the gases are exhaled through the exhaust opening in the valve, marked by the arrow.

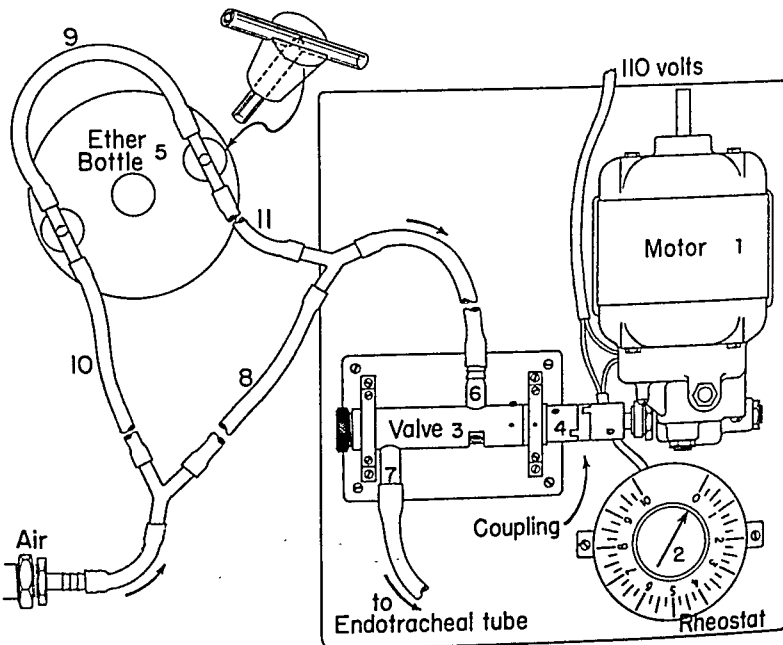


Fig. 2.—Diagram of apparatus. The numbers correspond to those in Fig. 1. (In place of the flexible coupling [4] shown here, one may use a "Multijaw Coupling," type FA 75, made by the Boston Gear Works, Inc., 14 Hayward St., North Quincy 71, Mass.)

STREPTOMYCIN PLUS ALKALINIZATION OF THE PLEURAL CAVITY IN THE TREATMENT OF TUBERCULOUS EMPYEMA

ENRIQUE STAINES, M.D., AND CONSUELO CÁRDENAS, M.D.

MEXICO CITY, MEXICO

WITH the advent of streptomycin to the therapeutic arsenal against tuberculosis^{30, 31} many hopes arose that it would be a great help in the management of tuberculous empyema. Indeed, the gratifying results obtained with this antibiotic in other localizations of tuberculosis, the advantages of penicillin in the local treatment of nontuberculous empyema (associated, of course, with sound principles of surgical drainage) made it reasonable to expect similar good results with streptomycin in tuberculous empyema. Disappointment and disillusion came with early reports; with the exception of Dobrie, Albertal, and Rodríguez Olivares,² it is a unanimous opinion that streptomycin is of little or no value at all in the treatment of tuberculous empyema.^{3, 4, 5, 9, 13, 15, 18, 19, 29, 31}

Such feelings are expressed by the Statements of the Committee on Therapy and Sub-Committee on Streptomycin of the American Trudeau Society: "Streptomycin therapy is not recommended for treatment of chronic empyema of tuberculous origin, because of its apparent ineffectiveness in studies thus far reported;"⁵ and by the last of the Reports to the Council on Pharmacy and Chemistry of the American Medical Association: "Chances for the successful control of tuberculous empyema with streptomycin therapy would appear to be exceedingly remote."⁴

Why has Streptomycin been ineffective so far, in the control of tuberculous empyema?

There are two pharmacologic and bacteriologic facts of extreme importance: First, streptomycin action is inhibited by an acid medium.^{1, 7, 8, 16, 18, 19, 20, 26, 27, 28, 31, 35} Second, tuberculous empyema has an acid reaction as has been proved by several investigators.^{10, 13, 11, 19, 33}

Neglect of those facts account, in our opinion, for the failures so far obtained, for nobody has reported attempts to reverse the acid reaction of tuberculous empyema during streptomycin therapy. Furthermore, in 1939, Canova and Agnello¹⁰ found out that if the empyema gets worse its pH goes down; but on the contrary, improvement of the empyema is paralleled by a tendency to alkalization of the pus. According to Trossarelli³³ in 1933, a pleural fluid with a pH above 8.1 inhibits the growth of tubercle bacilli in artificial media. These facts are of capital importance; the present treatment described by us has its foundations on them, associating, of course, sound principles of surgery for evacuation of the purulent material.

But there is another important factor in handling empyemas: broncho-pleural fistulas. Coryllos,¹¹ Auerbach,⁶ and Ornstein and Ulmar²² have explained the pathogenesis of tuberculous empyema in the course of intrapleural pneumo-

From the Chest Service of the Instituto Mexicano del Seguro Social.

Received for publication June 6, 1949.

DISCUSSION

Our usual anesthetic practice consists of induction by open ether for a few minutes, followed by introduction of an endotracheal catheter with an inflatable cuff. When the cuff is inflated, the respiratory exchange is carried on exclusively through the catheter. A convenient length of rubber tubing connects the catheter to the apparatus, which has been previously adjusted to a proper rate, air pressure, and concentration of ether. The respiratory rhythm is soon controlled by the machine, and the only subsequent adjustments involve shifting of clamps on the tubing to vary the depth of anesthesia. It may be necessary to add ether during prolonged procedures. At the completion of operation the catheter is disconnected from the apparatus to allow resumption of normal respirations before removal of the endotracheal tube. Spontaneous breathing is resumed usually in a minute or less. The machine provides satisfactory anesthesia in dogs, cats, or monkeys.

and 3 were bronchoextrapleural fistulas (B.E.F.). Three patients had cutaneous-extrapleural fistulas (C.E.F.).

DEFINITION OF CRITERIA

Complete cure of the empyema is considered here when there was total disappearance of fluid, when the cavity became sterile and bronchial fistula was demonstrable no more. Improvement of the empyema is accepted when at the end of the treatment fluid in small amount is still present, but it must be serous in character and negative for acid-fast bacilli in repeated examinations; patients with dry, sterile cavity but in whom a fistula is still present at the end of the treatment are considered as improved. The empyema is labeled unimproved when Koch bacilli are still present in the fluid at direct examination, no matter if there is general improvement and the fluid changes in physical characteristics; cases with purulent, sterile fluid are considered as unimproved.

Criteria for improvement and unimprovement might seem rather severe, but any new procedure or technique must be tested rigidly.

If parenchymal lesions are under satisfactory control by pneumothorax, but not completely arrested, the pneumothorax is maintained. In those with arrested lesions, the lung is allowed to re-expand. When the pneumothorax is doing no good for the pulmonary lesions, the program and form of collapse therapy is changed after healing or improvement of the empyema takes place. Patients in whom bronchial fistula is still present after streptomycin therapy will be submitted to surgical treatment.

RESULTS

Cured (23 patients).—Complete sterilization of the cavity and total disappearance of fluid was accomplished in all. Ten had extrapleural empyemas and 13 intrapleural empyemas. Two had B.E.F., 9 B.I.F., and 1 C.E.F., all proved at the onset of treatment; all were closed at the end.

Improved (10 patients).—In 8, a small amount of serous fluid, negative for acid-fast bacilli, was still present at the end of treatment; in fact, they are residual sterile exudates. The other 2 patients, both have their cavities completely dry and negative, but treatment failed to close their bronchial fistulas; 1 is B.E.F. and the other is B.I.F.

Unimproved (2 patients).—Both had fluid at the end of the treatment. One of them has a persistent cutaneous-extrapleural fistula provoked by repeated tappings at the most dependent site of the empyema, done elsewhere; this patient still has purulent fluid but negative for acid-fast bacilli. The other

TABLE I

NUMBER OF PATIENTS		PER CENT
Cured	23	65.7
Improved	10	28.6
Unimproved	2	5.7
Worse and deaths	0	0.0
Total		94.3
Good results		5.7
Unchanged		

thorax by the occurrence of bronchopleural fistulas. Their works are widely known and need no further comment. For correction of fistulas, parenteral administration of streptomycin is the logical step; closure of fistula and sterilization of caseous foci ruptured into the pleura can be accomplished, provided that (a) the fistula be recent, small, and with no fibrotic mouth, and (b) the affected lung be maintained collapsed during the treatment.

With the preceding considerations as a basis, an original technique for the treatment of tuberculous empyema has been developed; we have employed it since December, 1947.

TECHNIQUE

For evacuation of the pus by pleural tapping, we prefer an anterior approach, high up, through the first or second intercostal space at the level of the middle clavicular line, with a fine trocar, sufficiently long to reach the posterior wall of the chest and having the patient in dorsal decubitus with slight Trendelenburg position. By proceeding in this fashion, total evacuation of the pus can be accomplished, for the tip of the trocar reaches the bottom of the fluid; the trocar tract is not continuously bathed by pus afterward, so chances of fistulization are greatly diminished. All this, of course, is when there is an intrapleural or extrapleural pneumothorax space as there has been with all our cases. After local anesthesia of the skin and intercostal space is established with 1 per cent Novocain solution, a long trocar, No. 16, is inserted into the pleural cavity, the sharp portion is withdrawn, and the cannula gently pushed until it reaches the posterior wall of the chest. Aspiration of the pus is done with a syringe. When the cavity is empty, a pleural lavage with generous amounts of normal saline is done until the fluid comes back completely clear. Then, $\frac{1}{2}$ to 1 Gm. of streptomycin dissolved in 15 c.c. of 25 per cent sodium citrate solution is injected into the pleural cavity and the cannula is taken out.* If there is mixed infection (as there was in 5 cases), 100,000 to 200,000 units of penicillin are introduced along with the streptomycin-sodium citrate solution; the associated germs disappeared very soon, even in cases of putridity (2 cases).

To promote healing of fresh, soft, small bronchopleural fistulas and to sterilize caseous foci ruptured into the pleural cavity, parenteral streptomycin is administered along with the local treatment; we have employed low doses of 0.50 Gm. every twenty-four hours. Pleural permeability to streptomycin persists even in cases of thick pleura.^{17, 32, 36}

Collapse therapy is maintained until total disappearance or improvement of the empyema occurs. Then if it is considered helpful for the parenchymal disease, it is prolonged and maintained; or it is changed or discontinued according to the status of the pulmonary lesions.

MATERIAL

The material herein presented consists of 35 patients of both sexes, all adults, afflicted with tuberculous empyema; all under collapse therapy; the fluid was frank pus in all cases and Koch bacilli were found by direct examination in every instance. The age of the empyemas ranged from one to three months except in 1 case, 5 months old with wide, open, rigid bronchoextrapleural fistula.

Twenty empyemas developed during intrapleural pneumothorax therapy and 15 arose as complications of extrapleural pneumothorax. Bronchial fistulas were demonstrated in 15 patients; 12 were bronchointrapleural fistulas (B.I.F.)

*At the present time we are using a buffer solution with a high pH, to dissolve the streptomycin for local instillation. The pleural lavage is done also with strong alkaline solution in order to reverse the pH of the pleural cavity quicker.

Table III shows results according to closure of fistulas. We believe that most cases of tuberculous empyema developed during intrapleural pneumothorax are due to bronchopleural fistulas and discharge of caseous material inside of the pleural cavity. This is not quite as universal for extrapleural pneumothorax complicated with empyemas. As tests for the demonstration of bronchial fistulas, injection of methylene blue and mint essence as well as careful recordings of manometric pressure on pleural cavity were done. This last one gave in our hands the most consistent results. Unfortunately, we did not perform gasometric studies which comprise the best method.

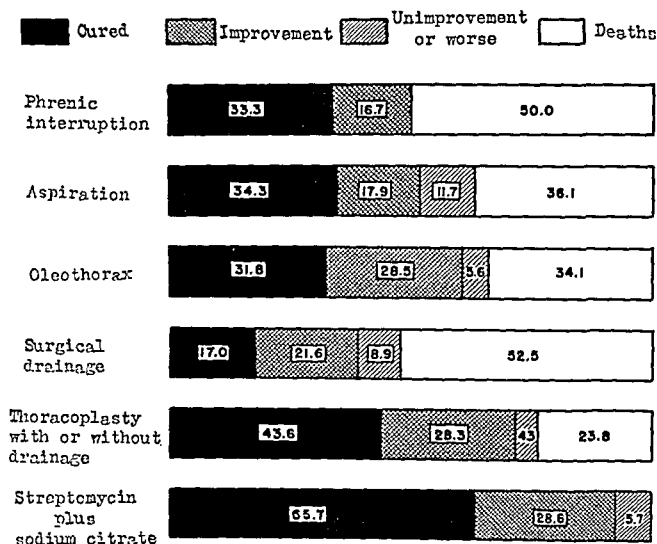


Fig. 1.—Comparative results. The percentages, except the streptomycin figures, were collected by Ehlers in a review of the cases reported from 1930 to 1941 by Hedblom, Jones, and Alexander, Skavlen, Woodruff, Berry, Dumarest, and Rougy, Peters and associates, Sayago and Lastra, etc.

Bronchial fistulas were demonstrable in 15 instances; 12 were B.I.F. and 3 B.E.F. Thirteen bronchial fistulas were closed at the end of treatment; 11 were B.I.F. and 2 B.E.F. Important facts for success are: age of the fistula and pathologic status of the opening. The 2 patients (1 B.I.F. and 1 B.E.F.) on whom treatment failed to close the fistulas had wide open fibrotic, old bronchial mouths. There were 3 cases of extrapleural-cutaneous fistulas. All reached us with their tracts already established; 1 carrying tube drainage and the other 2 with fistulized needle tracts, for they had been tapped several times elsewhere, at the most dependent place of the empyema. Two tracts closed spontaneously when the empyema disappeared and 1 is still open (Fig. 1).

is a patient with silicotuberculosis; intrapleural pneumothorax was performed on him in order to stop repeated massive hemoptysis; so far, the pneumothorax has accomplished its hemostatic purpose but has failed to control the lung lesions; this patient still has some fluid in his pleural cavity, serous in character and positive for acid-fast bacilli.

TABLE II. FORMS OF COLLAPSE DURING AND AFTER TREATMENT

NUMBER OF PATIENTS	
pneumothorax maintained	22
	13 Intrapleural 9 Extrapleural
Changed to oleothorax	6
	3 Intrapleural 3 Extrapleural
Lung purposely re-expanded	3
Waiting for other procedures	4
	Arrested lesions

Table II shows the type of collapse during treatment and its outcome. The status of pulmonary lesions is the crux of such a matter. In 22 cases it was considered advisable to maintain the pneumothorax space because it was exerting beneficial influence upon the lung lesions, although this was not yet totally arrested. Thirteen of such patients have intrapleural pneumothorax and 9 have extrapleural ones; 17 have their empyemas cured and in 5 they are improved.

Six patients were changed to oleothorax (3 intrapleural and 3 extrapleural); in all of them it was considered necessary to maintain the collapse, but all showed a tendency to develop symphysis after treatment of the empyema; in order to maintain the efficient space, mineral oil was used, only as an anti-symphysary measure. Five have their empyemas cured and 1 improved.

In 3 patients the lung was allowed to re-expand after healing of the empyema occurred. All had long-standing collapse with arrested pulmonary lesions; all have been under control for more than five months and their status is quite satisfactory for both pleural and pulmonary status.

There are 4 patients waiting for other procedures to be done. Two patients are improved and have patent fistulas, on them, thoracoplasty will be done. Two other patients are unimproved ones: one has a cutaneous-extrapleural fistula and the other is a silicotuberculosis patient. On the first, direct surgical attack upon the fistula is being planned; on the other, we are perplexed about what to do, for we are completely pessimistic about the outlook for any form of collapse therapy on silicotuberculosis patients.

TABLE III. INFLUENCE OF THE TREATMENT ON FISTULAS

	BEFORE TREATMENT	AFTER TREATMENT	
	PATENT	CLOSED	OPEN
Bronchointrapleural	12	11	1
Bronchoextrapleural	3	2	1
Cutaneous-extrapleural	3	2	1
Total	18 (100%)	15 (83%)	3 (17%)

3. *Ubi Pus Vacua*: The fulfillment of this old and wise principle of general surgery is observed by evacuation of the pus through a safe approach to avoid fistulization.

The beneficial influence of the treatment is reflected two ways, local and general. Locally, the fluid thins out, becomes serous, the amount decreases, its reaction reverses to alkaline, and Koch bacilli disappear soon. The general gain parallels local improvement: fever subsides, patient gets appetite and strength, and weight improves. There were no signs of intolerance for the streptomycin and no patient became worse or died in the present series.

The excellent results obtained by this method can be compared with advantage with other types of treatment for tuberculous empyema (see Fig. 1). Among 35 patients, 23 were cured (65.7 per cent). Ten patients were improved (28.6 per cent); in eight of them, the effusion is not a problem anymore; 2 other patients still have bronchial fistulas, but sterile, empty empyema cavities. It can be fairly said that in 94.3 per cent of the patients good results were obtained. This is in a very sharp contrast with the results of streptomycin therapy of tuberculous empyema obtained by the group of veterans hospitals as it was reported to the Council of Pharmacy and Chemistry of the American Medical Association;⁴ they reported 35 cases, too, and 22 of them were considered as unimproved (61.4 per cent). In their report, there is no mention of alkalinization of the pleural cavity.

SUMMARY

Failure of streptomycin for the treatment of tuberculous empyema is generally recognized. The causes of this failure are discussed. Four factors have capital importance:

1. Streptomycin action is inhibited by an acid medium.
2. Tuberculous empyema exudate has an acid reaction.
3. A high pH inhibits proliferation of Koch bacilli in pleural fluid.
4. Early parenteral streptomycin therapy may help to heal fresh, soft, small fistulas.

An original technique for treatment of tuberculous empyema is presented. This technique is based on the following: Alkalinization of the pleural cavity plus local instillation of streptomycin. Frequent removal of pus performed by pleural tapping through a high, anterior approach. Associated parenteral administration of streptomycin.

Advantages of this technique are enumerated, and results obtained in 35 cases of proved tuberculous empyema are presented.

The technique described gives better results than any other procedure employed up to now for treatment of tuberculous empyema.

Acknowledgment.—We are very grateful to Dr. Fernando Quijano-Pitman for his valuable help in the realization of our work and his assistance in the translation of this paper, to Dr. Alberto Monnier-Millote for doing the laboratory work in our cases, and to the staff of the Unidad de Neumología y Cirugía de Torax del Instituto Mexicano del Seguro Social and other colleagues who sent us their patients with tuberculous empyema to be treated by our method.

DISCUSSION

Since December, 1947, when we began to employ the treatment herein described, we have finished treatment in 25 cases of tuberculous empyemas. All had purulent fluid, positive for acid-fast bacilli. Most of them reached us with their empyemas already established and were kindly sent by other colleagues who knew our interest in the subject. Presence of fistulas was routinely investigated by intrapleural instillations of methylene blue and mint essence and by manometric studies of pressures of the empyema cavity. Gasometric studies were not done. We believe that the real number of fistulas was, in fact, greater, but many were too small, of valvular action, intermittently open, and not detectable by the methods employed by us. Ninety-one and seven-tenths per cent of B.I.F. were closed at the end of the treatment and 4 out of 6 B.E.F. and C.E.F. were closed, too (66.7 per cent). The frequency of aspirations varied widely and was regulated by the reproduction of the fluid; usually they were made with intervals of from four to ten days. We prefer by far to make repeated aspirations over the use of tube drainage on the most dependent site of the empyema, in order to obtain better control of the pneumothorax when it is indicated to maintain it and to avoid chest wall fistulas, as it was with one of our patients who came to us carrying his tube. Performing the aspirations through an anterior approach, high up, in the first or second intercostal space, far away from regions continuously bathed by recurrent pus, minimizes the risk of a chest wall fistulization by repeated tapping at the most dependent site of the empyema; we have 2 of such patients who came to us with fistulized needle tracts.

Our technique has its foundations in:

1. Sound bacteriologic and pharmacologic principles:

- (a) Streptomycin action is inhibited by an acid medium.
- (b) Tuberculous empyema has an acid reaction.
- (c) Pleural fluid with pH above 8.1 inhibits growth of Koch bacilli.

Therefore, alkalization of the pleural cavity must be done to create a favorable medium for streptomycin action and to make difficult the growth of Koch bacilli. This is accomplished by instillation of 25 per cent sodium citrate solution plus streptomycin into the pleural cavity*.

2. Pathologic facts: The pathogenesis of tuberculous empyema during intrapleural pneumothorax and sometimes during extrapleural pneumothorax is due to a bronchial fistula and discharge of caseous material inside the cavity. Early local attack and parenteral streptomycin before irreversible fibrosis has taken place are the logical steps for correction of this condition. Failure to close bronchial fistulas in this series is due to old fibrotic openings.

*The important principle of alkalization of the medium plus local streptomycin will have wider applications for the treatment of other forms of tuberculosis. Endobronchial segmental instillations of streptomycin plus sodium citrate solution into giant cavities of tuberculous origin, located at the posterior basal segment are being done by Dr. Quijano-Pitman²³ in 2 cases with quite satisfactory results, to improve the cavity bronchus condition. Of course, collapse therapy will be associated when the local condition will permit it. Dr. Quijano-Pitman also has injected transparently this solution into a giant cavity located on the posterior segment of the right upper lobe before thoracoplasty.

26. Rake, G., and Donovick, R.: Procedure for Testing Sterility of Concentrated Streptomycin Solutions, *Proc. Soc. Exper. Biol. & Med.* 62: 31, 1946.
27. Robinson, H. J., Smith, D. G., and Gaessie, O. D.: Chemotherapeutic Properties of Streptomycin, *Proc. Soc. Exper. Biol. & Med.* 57: 226, 1944.
28. Ruiz, Sanchez A.: Farmacologia de la Estreptomieina y su Aplicacion al Tratamiento de la Tuberculosis Pulmonar, *Rev. mex. de tuberc.* 8: 335, 1947.
29. Samson, P. C.: The Prophylactic Administration of Streptomycin Before and After Thoracic Surgical Operations, *Am. Rev. Tuberc.* 58: 38, 1948.
30. Schatz, A., Bugie, E., and Waksman, S. A.: Streptomycin: A Substance Exhibiting Antibiotic Activity Against Gram-Positive and Gram-Negative Bacteria, *Proc. Soc. Exper. Biol. & Med.* 55: 66, 1944.
31. Schatz, A., and Waksman, S. A.: Effects of Streptomycin and Other Antibiotic Substances Upon Mycobacterium Tuberculosis and Related Organisms, *Proc. Soc. Exper. Biol. & Med.* 57: 244, 1944.
32. Steenken, W., Jr., D'Esopo, N. D., and Wolinsky, E.: Excretion of Streptomycin Into Tuberculous Cavities, Pleural Space and Tracheobronchial Tree, *Am. Rev. Tuberc.* 56: 403, 1947.
33. Trossarelli, L.: Fenomeni di dissociazione del Bacterium Pyocianum, *Gior. di batteriol. e immunol.* 10: 800, 1933.
34. Veterans Administration, Army and Navy. Combined Report to the Council on Pharmacy and Chemistry of the American Medical Association: Effects of Streptomycin in Man, *J. A. M. A.* 135: 641, 1947.
35. Wolinsky, E., and Steenken, W., Jr.: Streptomycin and Penicillin Resistant Staphylococci; Influence of pH Body Fluids on Streptomycin Action, *Proc. Soc. Exper. Biol. & Med.* 62: 162, 1946.
36. Zintel, H. A., and Others: Studies on Streptomycin in Man: Absorption, Distribution, Excretion and Toxicity, *Am. J. M. Sc.* 210: 421, 1945.

ADDENUM

A first report, including 30 cases, was delivered in Spanish by us at the Third National Congress of Tuberculosis and Silicosis held at Mexico City in January, 1949. Shortly thereafter, at the same Congress, Professor Attilio Omodei-Zorini from the Forlanini Institute of Rome, presented a paper in which he stated that on a date posterior to the initiation of our work and without previous knowledge of it, the Italian authors began to employ a method for the treatment of tuberculous empyema based on the same principles on which our method is founded. Their technique, however, differs slightly from ours, for they use permanent closed drainage by means of a tube inserted on the most dependent place of the empyema; they perform daily lavage and instillation of streptomycin plus sodium phosphate instead of sodium citrate. Up to January, 1949, they had treated 22 cases of tuberculous empyema with quite satisfactory results.

REFERENCES

1. Abraham, E. P., and Duthie, E. S.: Effect of pH of Medium on Activity of Streptomycin and Penicillin and Other Chemotherapeutic Substances, *Lancet* 1: 455, 1946.
2. Albertal, M., Dobric, L. L., and Rodriguez, Olivares J.: Experiencia Clinica en el Tratamiento de la Tuberculosis por la Estreptomicina, *Rev. panam. med. y cir. tórax* 1: 235, 1948.
3. American College of Chest Physicians. Subcommittee on Chemotherapy and Antibiotics: Streptomycin. A Report on Its Present Use in Tuberculosis, *Dis. of Chest* 13: 169, 1947.
4. American Medical Association. Council on Pharmacy and Chemistry: Streptomycin in the Treatment of Tuberculosis Current Status, *J. A. M. A.* 138: 584, 1948.
5. American Trudeau Society. Committee on Therapy and Subcommittee on Streptomycin: Report, *Am. Rev. Tuberc.* 56: 766, 1947.
6. Auerbach, O.: Personal communication.
7. Berkman, S., Henry, R. J., and Housewright, R. D.: Studies on Streptomycin, *J. Bact.* 53: 567, 1947.
8. Bloch, H., Matter, E., and Suter, E.: Effect of Glycerol and Related Substances on Growth and Oxygen Uptake of Tubercle Bacillus, *Am. Rev. Tuberc.* 55: 540, 1947.
9. Bogen, E.: Streptomycin Treatment of Tuberculosis (Editorial), *Am. Rev. Tuberc.* 56: 442, 1947.
10. Canova, S., and Agnello, V.: Alcuni caratteri fisici e chimici dell'empima tbc. studiati anche a varia altezza, *Ann. Ist. Carlo Forlanini* 3: 232, 1939.
11. Coryllos: Cited by Ehler.
12. Coryllos, and Goldberg: *Fisiología Patológica de la Tuberculosis*, Tuberculosis Clinica, Goldberg and others, Barcelona, 1942, The University Society, Inc.
13. Ehler, A. A.: Tuberculosis Empyema; Collective Review of Literature From 1930 to 1941, *Internat. Abstr. Surg.* 74: 97, 1942.
14. Farber, S. M., and Eagle, H. R.: El Tratamiento de la Tuberculosis por la Estreptomicina, *Rev. panam. med. y cir. torax* 1: 279, 1948.
15. Freedlander, B. L., and French, F. A.: Constitution of Chemotherapeutic Agents in Experimental Tuberculosis, *Am. Rev. Tuberc.* 56: 373, 1947.
16. Geiger, W. B., Green, S. R., and Waksman, S. A.: Inactivation of Streptomycin and Its Practical Applications, *Proc. Soc. Exper. Biol. & Med.* 61: 187, 1946.
17. Heilman, D. H., and Others: Streptomycin: Absorption, Diffussion, Excretion and Toxicity, *Am. J. M. Sc.* 210: 576, 1945.
18. Hinshaw, H. C., and Feldman, W. H.: Streptomycin in Treatment of Clinical and Experimental Tuberculosis, *Ann. New York Acad. Sc.* 48: 175, 1946.
19. Hinshaw, H. C., Feldman, W. H., and Pfuetze, K. H.: Treatment of Tuberculosis With Streptomycin, *J. A. M. A.* 132: 778, 1946.
20. Notas Terapeuticas: El Valor Clinico de la Estreptomicina (Editorial), 41: 165, 1948.
21. Omodei-Zorini, A.: Estado Actual de la Lucha Contra la Tuberculosis en Italia, Paper Read at the Third National Congress of Tuberculosis and Silicosis at Mexico City, January, 1949.
22. Ornstein, G. G., and Ulmar, D.: Tuberculous Empyema: Classification, Pathogenesis and Treatment, *Quart. Bull., Sea View Hosp.* 4: 269, 1939.
23. Pfuetze, K. H., and Ashe, W. M.: Present Status of Streptomycin in Tuberculosis, *Dis. of Chest* 14: 446, 1948.
24. Pulaski, E. J., Kleinman, A. M., and Duncan, G. G.: Some Considerations of Clinical Application of Streptomycin, *Ann. New York Acad. Sc.* 48: 183, 1946.
25. Quijano-Pitman, F.: Personal communication.

This short paper is based on the experience of seventy-two complete thoracoplasties without a single case of infection, either deep or superficial. In all these cases the lower one-half of the scapula was resected prior to resection of the ribs and early bedding of the remaining scapula was obtained after the first stage.

In sixty-three cases the scapula was resected through a small incision along the vertebral border of this bone. Following the resection of one-half of the scapula, it was found that the trapezius and rhomboids need only be partially divided, as the direction of the scapula retraction was toward the shoulder instead of toward the axilla when the bone is intact. The latissimus and the serratus were spared altogether. In some cases the division of trapezius and rhomboid was so limited that the operation could have been termed a "pseudo-Finochietto thoracoplasty." It is granted that the exposure was not a very "comfortable" one, but in thin patients it was quite sufficient.

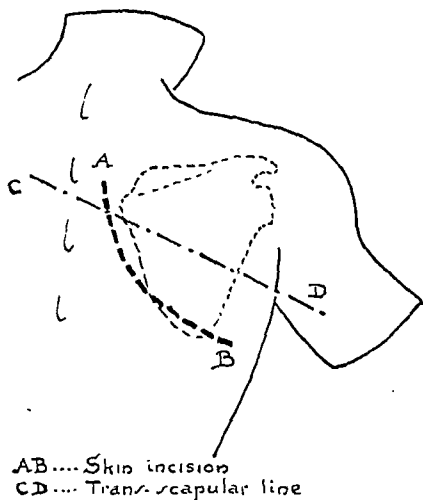


Fig. 1.

As is always the case with surgical techniques, I have found now that the rhomboid need not be divided at all, and even the trapezius can almost be spared, except for a small incision across its scapular edge, if the operation is performed through the transscapular approach that I am about to describe. As can be seen in the accompanying illustrations the technique is quite simple.

The skin incision can be a limited periscapular one, starting between the spine and the superior angle of the scapula, round this bone, to its inferior angle (Fig. 1). The skin flap overlapping the scapula is then undercut until the lower one-half of this bone is freed all over (Fig. 2). The infraspinatus is incised along

TRANSSCAPULAR THORACOPLASTY

ALFRED J. COELLO, M.D. (BARCELONA)

BARROW-IN-FURNESS, ENGLAND

THIS subject, unorthodox division of muscles in thoracoplasty, may not be considered by many surgeons as an important or interesting one. Nevertheless, several papers have been written on it, some of them quite original, like: "Thoracoplasty Without Section of Muscles" by Finochietto and "A Musculo-plastic Incision for Posterior Thoracoplasty" by Brock. Head and Wangensteen have also performed this operation through small incisions with splitting of muscles without dividing them. All these authors aimed at reducing the post-operative reaction, minimizing the impairment of shoulder movement and the amount of pain, and even bringing down the incidence of wound infection.

Wound infection in thoracoplasty operations is not so uncommon as it is generally believed. As Wangensteen said long ago, "the leaving of a 'dead space' which must fill with blood and sanguineous transudate invites infection and is admittedly in conflict with the well-established principle of obliterating all planes in which fluid may collect." According to Overholt and Betts, "the reports from the literature indicate that some degree of wound infection occurs in approximately 15 per cent of all cases." Gale and Oatway reported 18 per cent of infections in their 1936 series and only 9.5 per cent in their 1937 series. No doubt, with the great advances accomplished in thoracic surgery in the last years, the incidence of wound infection in thoracoplasty is considerably lower than it used to be.

In my paper, "Scapulectomy and Thoracoplasty," I tried to prove that infection of the extrapleural space can easily be avoided by following the advice given by Wangensteen, that is to say, by the early obliteration of that space. This can be accomplished by the resection of the lower one-half of the scapula during the first stage.

Wangensteen's advice to fill the extrapleural space with a synthetic rubber sponge coated with paraffin, like other procedures recommended for the obliteration of the "dead space" with different materials, had to be given up on account of a high incidence of failures.

In 1947 I thought that we had found the answer to extrapleural infection by the prophylactic use of penicillin-sulfathiazole powder, sprinkled over the operative area before the closure of the wound. Two cases of deep sepsis, occurring after the publication of my paper, have strengthened even further my conviction that the best and surest method of avoiding infection of the extrapleural space is by the early obliteration of the "dead space," by the apposition of the walls of this space, particularly its posterior and anterior walls. Anteriorly we know that the infraclavicular fossa will "sink" immediately after the resection of the anterior portions of the first three ribs. It is only the presence of the scapula that will keep a "dead space" over the collapsed lung.

can be finally divided with any straight bone cutter (Fig. 5). Once loose, the lower one-half of the scapula is gripped with a bone forceps and pulled out vigorously, therefore facilitating the subperiosteal dissection of its anterior aspect

Fig. 6.

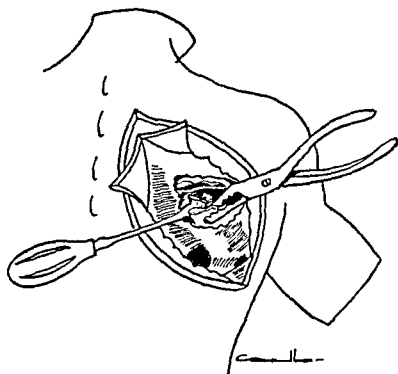


Fig. 7.

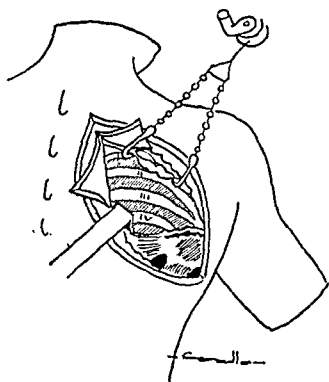
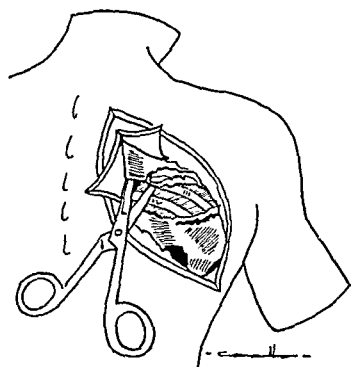


Fig. 8.

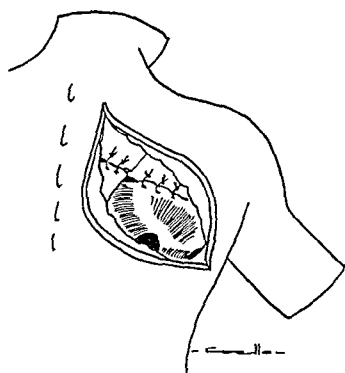


Fig. 9.

(Fig. 6). An opening is then made through the fibers of the rhomboid along a line in continuation with the transscapular incision. The tough periosteal bridge between the rhomboid and the subscapularis at this point has to be divided with

an oblique line roughly following the direction of the underlying fourth rib (Fig. 3). Through this opening the bone is freed subperiosteally. A tunnel is then made with the raspatory between the scapula and the subscapularis, wide

Fig. 2.

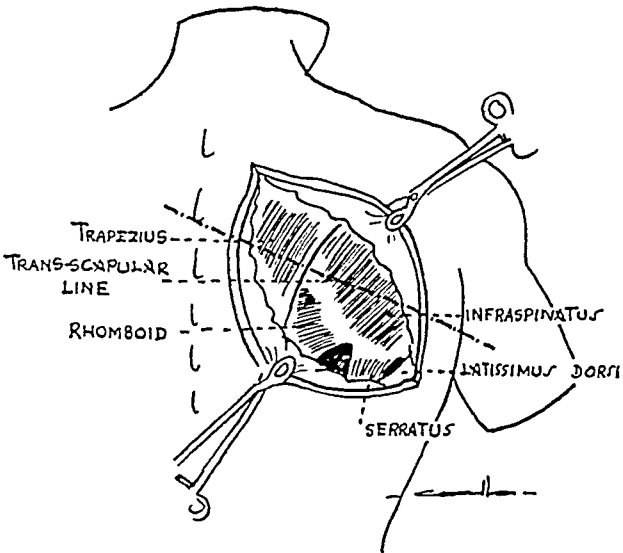


Fig. 3.

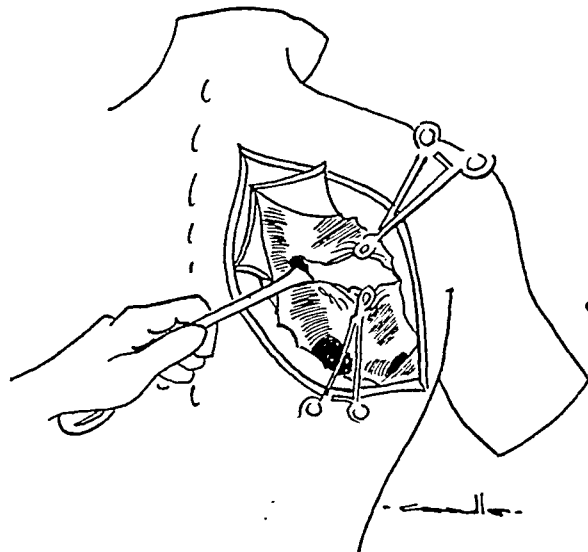
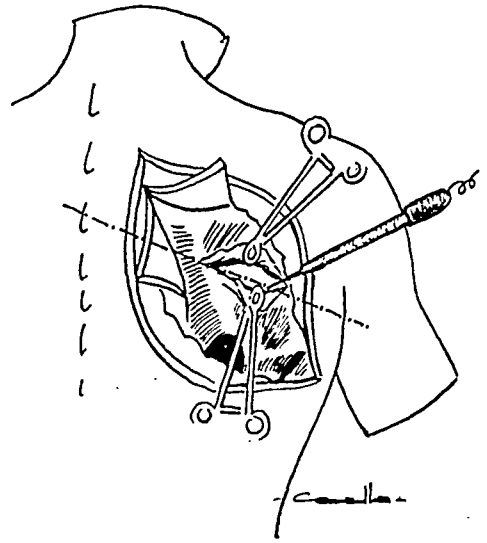


Fig. 4.

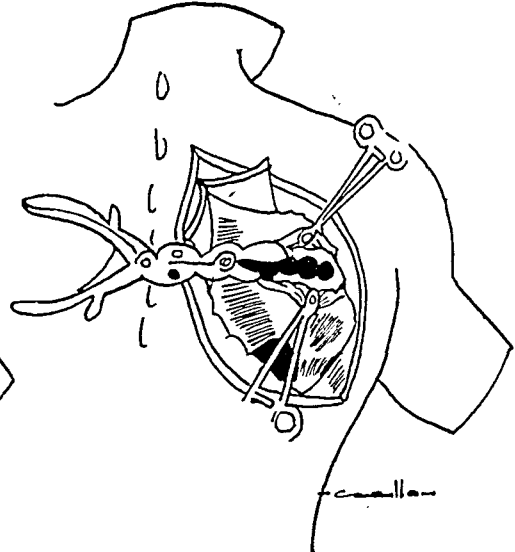


Fig. 5.

enough to allow later on its division with the bone rongeur (Fig. 4). No other bone cutter at our disposal can do this job so neatly and quickly. The bone is divided by successive bitings. The end bridge at the axillary border of the scapula

rhomboid need only be split along its fibers. The operation can be done in a short time, the amount of trauma is minimal, the postoperative recovery is very quick, and above all, it is an excellent prophylactic measure against extrapleural space infection due to the early obliteration of that space.



Fig. 11.

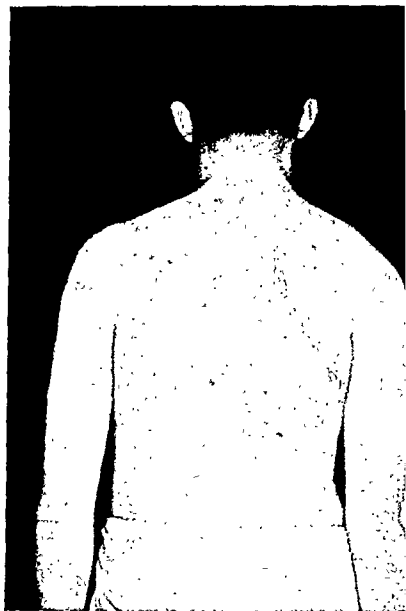


FIG. 12.

REFERENCES

- Finochietto, R.: Thoracoplasty Without Section of Muscles, *J. THORACIC SURG.* 9: 450, 1940.
 Brock, R. C.: A Musculoplastic Incision for Posterior Thoracoplasty, *J. THORACIC SURG.* 15: 182, 1946.
 Head, J. R.: Extrapleural Thoracoplasty, *J. THORACIC SURG.* 8: 55, 1938.
 Wangenstein, O. H.: Thoracoplasty for Tuberculosis and Chronic Empyema Through Short Incisions, *J. THORACIC SURG.* 8: 66, 1938.
 Overholt, R. H., and Betts, R. H.: A Comparative Report on Infection of Thoracoplasty Wounds, *J. THORACIC SURG.* 9: 520, 1940.
 Gale, J. W., and Oatway, W. H., Jr.: Indications, Hazards, and Results of Apicolysis Thoracoplasty, *J. THORACIC SURG.* 8: 78, 1938.
 Coello, A. J.: Scapulectomy and Thoracoplasty, *Brit. J. Tuberc.* 41: 75, 1947.
 Coello, A. J.: The Extrapleural Space in Thoracoplasty, *Tubercle* 27: 202, 1946.

It is seldom that one encounters marked symptoms from carcinoma of the lung until secondary infection has intervened. It appears that there is a decreased liability to early infection in tumors arising in the upper lobe areas. When a study is made of the localization of primary carcinoma, it is found that from 40 to 45 per cent of the lesions arise in an upper lobe. In a recent study made in our clinic of 112 primary carcinomas of the lung 51 of the lesions were found to arise in an upper lobe.

Fig. 1 signifies the proportion of patients found to deserve exploratory thoracotomy and the percentage of those justifying resection, in an over-all total of 2,731 patients derived from reports from the literature. The second, and similar column, signifies the same factors in 112 personal cases.

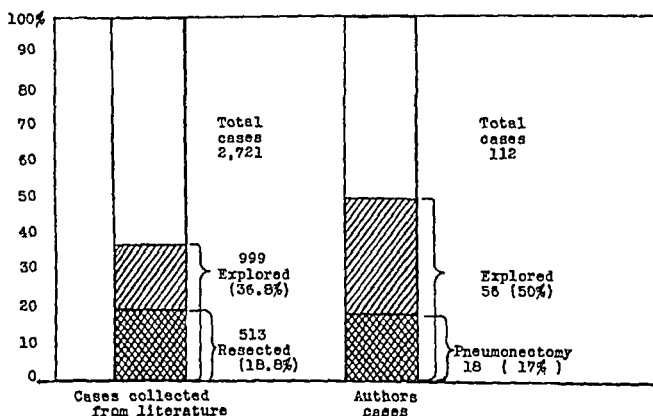


Fig. 1.—Graphic representation showing the existing frequency of inoperable primary pulmonary carcinomas. Incidence of explorations and operability in series from literature and from this series.

Fig. 2 is presented to show the localization of the primary site of the tumor in the 112 patients which the author has described elsewhere.¹ Our experience has been very similar to that of other investigators in that approximately 50 per cent of the patients in whom a diagnosis of carcinoma is made are found to be in an inoperable condition. In both our experience at Emory University and a study of the figures made at the Barnes Hospital, St. Louis,² we have found

Right upper lobe	30	Left upper lobe	21
Right middle lobe	4	Lingula	3
Right lower lobe	23	Left lower lobe	21
Right apex	4	Left apex	2
Right peripheral	6	Left peripheral	6
Total	67	Total	53

Fig. 2.—This chart records the localization of the primary neoplasm in 112 proved cases of primary pulmonary carcinoma. Particular stress is placed upon the primary lesion arising in the upper lobes in 54 of the total cases, or 42.8 per cent.

EXPERIENCES WITH THE SURGICAL RESECTION OF THE HUMAN CARINA, TRACHEAL WALL, AND CONTRALATERAL BRONCHIAL WALL IN CASES OF RIGHT TOTAL PNEUMONECTOMY

OSLER A. ABBOTT, M.D.

ATLANTA, GA.

IT HAS been a common unfortunate experience for physicians to discover carcinoma of the right upper lobe in such an advanced stage of its existence as to be felt beyond the help of surgical attempts at cure. In an over-all survey of the literature and personal cases with carcinoma of the lung totalling 2,843 patients, approximately 50 per cent of the patients are found to have the disease beyond the confines of the involved hemithorax, and therefore are considered inoperable. Of the remaining 50 per cent of the patients, in whom exploratory thoracotomy is felt to be justified, 36 to 60 per cent of those explored are found to have the lesion still in a resectable condition. Therefore, in an over-all summary of case reports found in the literature one may expect to find from 18 to 30 per cent of the patients, in whom a diagnosis of primary carcinoma of the lung is made, to have the disease in a stage satisfactory for one to feel that total pneumonectomy is justified with hope for eradication of the disease.

The accepted explanation for this remarkably poor outlook for carcinoma of the lung is felt to be due to three main factors. The first factor is the very insidious course of carcinoma of the lung in regard to early symptomatology and its marked ability to imitate other less serious lesions in the pulmonary system. The second factor has been the delay caused by the patient, consisting of the interval from the onset of symptoms to consultation with his physician. An inadequate consciousness of the possibility of this disease by the laity may therefore be considered to exist. The third and final factor has been the delay consequent to the physician, from the time of patient consultation to the making of a probable or positive diagnosis of carcinoma.

Certain new technical advances toward earlier positive diagnosis have recently appeared. These consist of (1) the study of the sputum and bronchial secretions for possible neoplastic cells, and (2) the increasing use of screening x-ray programs. As a result, these patients are being brought to the attention of the thoracic surgeon at an increasingly earlier stage of their disease. Certainly this has been true of lesions arising in the lower lobes. However, primary carcinoma in either upper lobe is still remarkably liable to be seen in a late stage in the course of the disease. This is considered to be due to two fundamental factors, namely, the marked tendency to consider an upper lobe carcinoma to be a tuberculous lesion, and an apparent tendency for upper lobe lesions to become symptom-producing at a later stage in the disease than that which is seen with lower lobe tumors.

From the Emory University School of Medicine, Department of Surgery (Thoracic).

Read (by title) at the Twenty-eighth Annual Meeting of The American Association for Thoracic Surgery, Quebec, Canada, May 31, 2, 1948.

have a similar concept of the potential danger of a large open area in the stump of the main-stem bronchus or even in the presence of a large area of excision of the lateral wall of the trachea and carinal region. It is our interpretation, based upon the fundamental concepts portrayed by the empyema commission,⁶ that adequate aeration of the remaining lung may be obtained as long as two factors are considered, namely, the maintenance of the mediastinal structures in their midline position, and the obtaining by the remaining lung of adequate amounts of oxygen.

It has been the policy of our clinic to support the mediastinum by traction sutures either in the wall of the involved bronchial stump, the trachea, or surrounding mediastinal structures whenever an open bronchial stump is produced. Emphasis is placed upon adequate suction measures in the region of the open stump, to prevent the spillage of blood or other materials into the functioning tracheobronchial tree. Furthermore, at the time during which the bronchus is opened, special suction tips are introduced to allow adequate clean-out of the tracheobronchial tree whether one is performing a pneumonectomy or a lobectomy. We have been impressed with the marked value of this procedure and its complete freedom from danger. The suction maneuver is carried out with due consideration of the removal of oxygen which is being delivered to the patient so that the actual time interval of suction is limited at all times to periods of thirty seconds.

We have also found clinical confirmation of the concept indirectly presented in this regard by the empyema commission, relative to the amount of paradoxical or disturbed respiratory function which may be permitted in relation to the patient's vital capacity. Thus, in a patient with a marked decrease in function of the contralateral lung, it may be necessary to introduce the endotracheal tube past the area of open trachea into the main bronchus. Furthermore, a patient may have a potentially high vital capacity of the remaining lung tissue markedly disturbed by aspiration of material spilled over from the resected, diseased lung tissue. Adequate aspiration through the tracheal defect will then permit withdrawal of the endotracheal tube and oxygen delivery so long as the mediastinum is supported. It should be mentioned that this operation is only performed with the patient supine. An anterolateral incision is utilized whenever tracheal or carinal region is planned or considered a possibility.

In all patients undergoing this procedure, a very marked physiologic disturbance characteristic of anoxia could be produced by relaxation of the stabilization sutures in the mediastinum prior to closure of the tracheobronchial defect. It is felt that this stabilization allows the intrapleural pressure in the left hemithorax to become adequately negative in relation to atmospheric pressure to allow adequate aeration of the left lung. As this mediastinal support is relaxed there ensues a decreasing expansion of aerating left lung with resultant progressive anoxia. In corollary fashion overdisplacement of the mediastinum toward the right will decrease the efficiency of expiration. Therefore, these stabilization sutures are retracted only to maintain the persisting portions of trachea and left main-stem bronchus directly above the dorsal vertebra. With this in mind, there is considerably less chance for these traction

the highest proportion of inoperable cases to consist of patients with upper lobe lesions. Thus, inoperable lesions were considered present in from 70 to 80 per cent of upper lobe tumors.

A further subdivision of these lesions has shown an even greater tendency for lesions arising in the right upper lobe to present themselves in an inoperable condition. The apparent reason for this higher inoperable tendency has been considered due to two main causes, namely, the proximity of the superior vena cava to the primary lesion, as well as the frequency with which right upper lobe lesions travel upward along the right lateral tracheal wall, and/or involve the carina and medial wall of the contralateral bronchus.

Claggett and his co-workers³ presented a very detailed and valuable study showing the marked tendency for bronchiogenic carcinoma to travel in a cephalad direction along the trachea and bronchus. He has suggested that one should obtain from 1.5 to 2.5 cm. of uninvolved bronchus proximal to a tumor if one is to hope to achieve eradication of this lesion.

The marked variance for the site of origin of the right upper lobe orifice has received considerable comment. Although the average normal position is described as 2.5 cm. below the carina, a much higher point of origin is frequently seen and occasionally the orifice may arise from the lateral wall of the trachea itself. Therefore, in view of the variable sites of origin of the right upper lobe orifice, as well as the marked tendency for lesions arising in the upper lobe to present involvement of the trachea or carina at the time of definitive diagnosis, it is considered that the technical considerations utilized in pneumonectomy, wherein a right upper lobe tumor is present, must of necessity differ from similar considerations wherein the primary lesion arises at a greater distance from the trachea and carina.

The purpose of this communication is to describe our personal experiences with an attempt to extend the indications for resection in right upper lobe carcinoma of the lung. Certainly one should not condone the need to have to carry out a considerably more hazardous and extensive resection, and the primary purpose of the men interested in this subject should be made toward an earlier diagnosis of the lesion before this type of complication is presented. Nevertheless, it is felt that there will be certain patients, even in the presence of an early diagnosis, in whom the position of the primary tumor will require the excision of tracheal wall, carina, and contralateral bronchial wall in order to obtain an adequate margin of healthy tissue beyond the primary lesion. The author also feels that the method which is to be presented will, in all probability, be discarded as the fundamental experimental work of Daniel⁴ achieves a safe application to the human being. However, in our experiences with this method, we have also encountered certain interesting physiologic and physiopathologic clinical observations which are felt to be worthy of report.

One frequently encounters statements in the literature indicating a fear on the part of the operating surgeon of the danger consequent to an open stump during the performance of total pneumonectomy. In a recent article by Strieter,⁵ in discussing the value of intracardiac adrenalin in cardiac resuscitation, this concept was again stressed. In our clinic we do not

We have adhered to the policy at the time of removal of a lung for carcinoma to obtain frozen sections upon the proximal stump of the bronchus of the resected lung. Should this reveal evidence of submucosal extension of the tumor, or direct examination of the resected specimen show that an inadequate margin of normal tissue has been removed, surrounding tracheal wall, carina, or contralateral bronchial wall is removed until such an adequate margin is felt to have been obtained.

With these principles in mind, our first patient to be considered in this discussion was encountered eleven months prior to the writing of this paper. In order, to eradicate the area of disease we were left with a large oval defect extending one inch up the right lateral tracheal wall, and the carina, together with a portion of the medial wall of the left main-stem bronchus, was removed. The cartilage on the lateral wall at the juncture of the trachea and origin of the left main-stem bronchus was found to produce a marked inward projection of the wall with consequent occlusion of the airway, when the trachea was held in the midline and an increasing degree of obstruction was produced as the mediastinum was drawn toward the right side. It was obvious, therefore, that the closure of this defect by a nonrigid substance such as a large pleural graft, would be unsatisfactory. Furthermore, such a graft could be compressed toward the left lateral wall of the tracheobronchial juncture with increasing pressure produced within the right hemithorax.

It was found that by mobilization of the tissues adjacent to this area, that the basic plastic surgical principle of a transverse closure of a longitudinal wound would both enlarge the lumen of the airway as well as tend to swing the left main-stem bronchus into a more vertical position (Fig. 4). The endotracheal tube was therefore advanced downward to the left main-stem bronchus and sutures laid, in order to accomplish this repositioning, and a satisfactory closure obtained. This defect was then covered with a pleural graft and no complications were encountered until the endotracheal tube was withdrawn up into the trachea and positive pressure introduced. When this was done the mediastinum tended to move laterally toward the right thorax and evidence of obstruction was encountered. As soon as the positive pressure was released and the mediastinum allowed to resume a position slightly to the left of the midline, an adequate airway was obtained. Therefore, with these considerations, when the patient's thorax was closed a slight positive pressure was maintained within the right hemithorax. In this first patient, because of a fear of the liability toward disruption of the suture line, the right pleural cavity was evacuated of fluid and a positive pressure pneumothorax maintained.

The patient underwent an uneventful recovery but it was noted during the postoperative course, that when the mediastinum shifted toward the right side and the pneumothorax allowed to become negative in both phases, dyspnea ensued. It was necessary, therefore, to maintain constant refills during the first six weeks postoperatively, after which time an increasing interval was allowed apparently due to fixation of the mediastinal structures. However, as time progressed there was a definite undesirable tendency for the mediastinum to assume a position to the right of the midline, which could not be satisfactorily

sutures to be pulled through the tissues by an overzealous assistant. These sutures are removed or in some places utilized in application of the pleural graft overlying the suture line or artificial trachea. Fig. 3 demonstrates the typical placement of these stabilizing sutures. In our experience when these stabilizing sutures are properly used, there is no danger inherent upon an open major bronchial stump or huge intrathoracic opening in the tracheal wall. Furthermore, no positive pressure need be delivered into the surviving lung nor direct instillation of oxygen unless there exists a markedly lowered vital capacity in that lung.

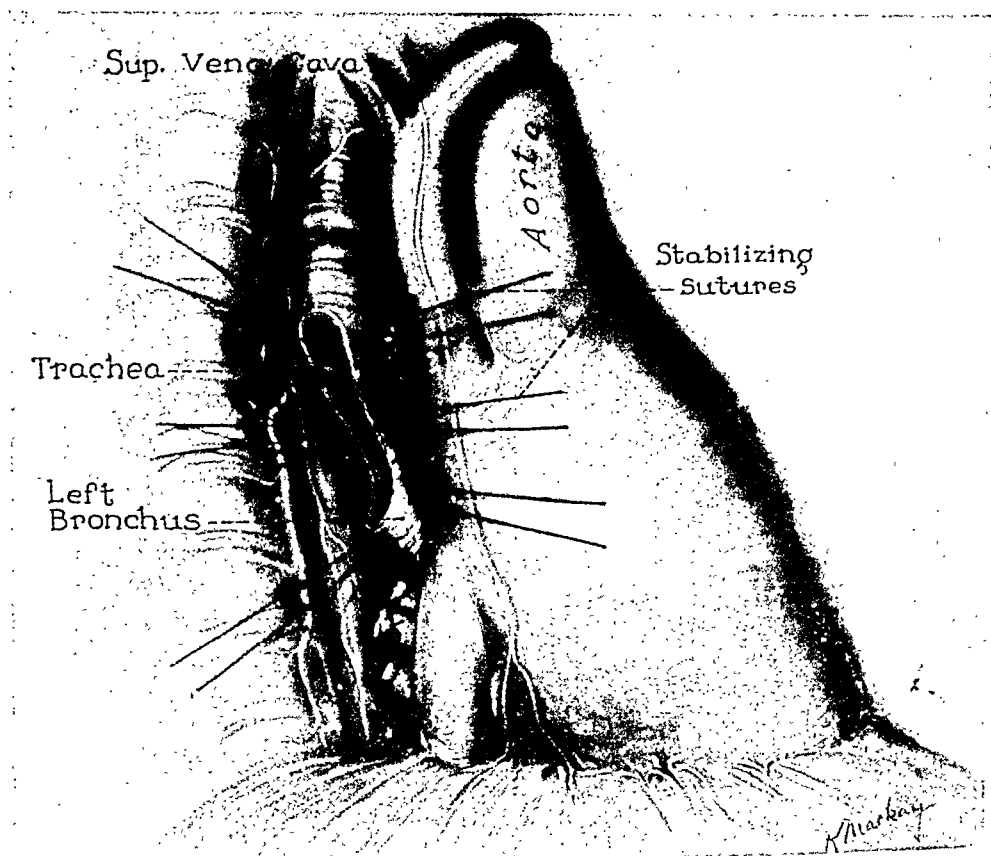


Fig. 3.—Artist's drawing demonstrating the placement of traction sutures used to stabilize the trachea and left main-stem bronchus in patients undergoing resection of these structures.

We have been sufficiently impressed with this basic concept that we have utilized it in the repair of large tracheoesophageal fistulas in newborn infants. Thus, in the newborn child in whom a large area of atelectasis and considerable soilage of the bronchial tree is presented and preoperative bronchoscopic measures are required, we support the posterior surface of the trachea prior to transecting the area of fistulous communication. After the area of fistula has been transected, a small catheter is introduced directly into each main-stem bronchus to clean out the bronchial tree. We have found this procedure to be of a definite benefit to the patient, and apparently without associated danger.

by an abdominal distention as well as an episode of acute gastric dilatation. Again these complications did not respond to Wangensteen suction alone, but required the maintenance of a positive pressure pneumothorax on the right. The patient was discharged from the hospital three weeks following surgery, but failed to return for pneumothorax refill and was admitted to the hospital in severe respiratory and abdominal distress three weeks later. Abdominal symptoms had been present for approximately five days and associated with

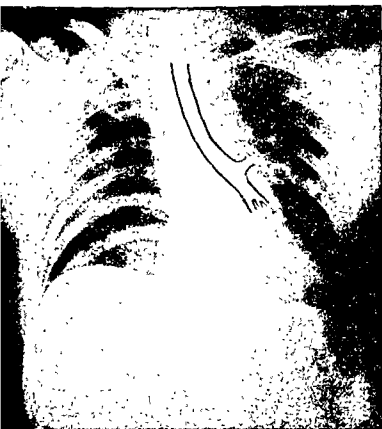


Fig. 12.



Fig. 13.

Fig. 12 (P. R.).—Portable x-ray film study of chest illustrating position of newly constructed airway after emergency right positive pressure pneumothorax. Left hilar shadow represents site of contralateral spread of tuberculosis. This patient had both carcinoma and active pulmonary tuberculosis in resected right lung.

Fig. 13 (P. R.).—Illustration of specimen of lower trachea and left main-stem bronchus obtained at autopsy 76 days following operation. Defect closed by transverse closure of longitudinal lateral elliptical defect. Satisfactory primary healing obtained. Two silk sutures visible at closure site appear loose and ready to be coughed up.

severe obstipation. A fecal impaction was removed, but adequate relief of symptoms was not obtained until reposition of the mediastinal structures had occurred. This patient was alive nine months following operation, without evidence of recurrence of disease.

A fourth patient (Figs. 12 and 13) with advanced primary right upper lobe carcinoma underwent similar operative procedure in December, 1947. No untoward difficulties were experienced at the operating table, but one hour following completion of the operation severe acute respiratory distress occurred, and it was found that the mediastinum had become markedly displaced toward the right, with considerable escape of air from the right pleural cavity into the

not available for direct visual examination or biopsy. A gastroenterostomy was performed, but following operation the patient persisted in marked pyloric obstruction and a nonfunctioning stoma with persistent nausea and vomiting. It was interesting to note that this nausea and vomiting persisted despite the institution of adequate Wangensteen suction. In view of this situation being noted prior to exploratory laparotomy, special attention was paid at that time to the possibility of a low "closed loop" obstruction. No evidence of any form of intestinal obstruction was discovered. It was felt necessary, therefore, to attempt to displace the mediastinum again to the left of the midline, which

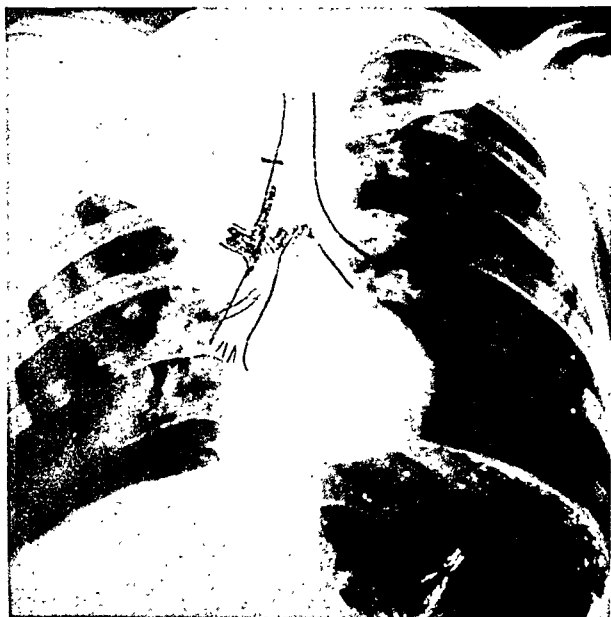


Fig. 10.



Fig. 11.

Fig. 10 (H. H.).—Posteroanterior x-ray film study showing complete atelectasis right upper lobe. Diagram insert illustrates degree of tracheal and carinal extension of the primary lesion. Note also the massive air shadow below left diaphragm produced by megacolon.

Fig. 11 (H. H.).—Postoperative x-ray film demonstrating position and outline of newly constructed airway following radical right pneumonectomy.

could not successfully be done with air, and plasma was injected into the right pleural cavity under considerable pressure. Again this repositioning of the mediastinum produced alleviation of the gastrointestinal symptoms and plasma has periodically been replaced in the thoracic cavity since that time. The patient was alive and well eight months following the original pneumonectomy, without evidence of recurrence of disease. It is our feeling that this patient presented persistent complete pyloric obstruction produced in some reflex fashion by faulty displacement of the mediastinum. This severe spasm did not respond to large doses of atropine or to Urecholine.

A third patient (Figs. 10 and 11) with advanced primary carcinoma arising in the right upper lobe with involvement of the carina and right lateral tracheal wall was operated upon in October, 1947. This patient presented the preoperative complication of a massive megacolon, which markedly displaced the left diaphragm upward. The patient's postoperative course was complicated

cinomatous tissue was found within the thorax. The exact cause of death in this patient is not explained, but is felt to be due probably to a summation of several factors, namely, the chronic pneumonitis in the left lung, the empyema in the right lung, and some destructive changes in the liver which might well have been produced by prolonged anoxia.



Fig. 14.

Fig. 14 (E. C.).—Posteroanterior view of chest showing obstruction of right upper lobe and main-stem bronchus. Tumor extended up the trachea and across the carina.



Fig. 15.

Fig. 15 (E. C.).—X-ray film of chest taken following extensive tracheal wall and left main-stem bronchial wall resection and radical right pneumonectomy. New airway constructed about a glass tube. This glass tube was essentially radiolucent.

It should be mentioned that in all the previously mentioned patients in whom this large area of tracheal reconstruction has been carried out, it has been our policy to institute an adequate-sized tracheotomy at the time of completion of the pneumonectomy. This was done in order to allow immediate access to the tracheobronchial secretions. It was also felt that the presence of a tracheotomy might decrease the pressure upon the suture line produced by strenuous coughing against a closed glottis. It is still our policy to carry out this routine tracheotomy with this procedure, and this was done in a sixth patient operated upon one month ago in whom a glass artificial trachea was again utilized (Fig. 16).

The actual salvage which may be derived from the application of this procedure to advanced right upper lobe carcinoma cannot as yet be evaluated. The first patient presented evidence of metastatic disease involving the upper portion of the right femur nine months following surgery, but is still alive with the disease at this time. The second and third patients were alive and

subcutaneous tissues. A bronchoscope was therefore inserted, noting marked kinking and obstruction of the trachea at the juncture of the trachea and the left main-stem bronchus on its left lateral wall. The patient experienced no difficulty as long as the bronchoscope was maintained in place but severe symptoms of anoxia immediately presented as soon as the bronchoscope was withdrawn up into the trachea. Finally a firm compression type of dressing was placed over the right chest and positive pressure pneumothorax instituted, allowing withdrawal of the bronchoscope and maintenance of an adequate airway. This patient was found to have both active tuberculosis and carcinoma in the removed right lung. A complete resection of mediastinal nodes was performed at the time of surgery and all these glands were found to be involved by metastatic disease. The patient had a stormy postoperative course, and finally appeared to be progressing well two months following operation when he developed signs of a transverse myelitis and died two weeks later. Autopsy revealed satisfactory healing of the repaired tracheobronchial juncture, no evidence of persistent malignant disease within the thorax. Very extensive metastatic disease was found about the entire thoracic portion of the spinal cord as well as diffuse metastatic lesions throughout the liver and peritoneal cavity.

The fifth patient (Figs. 14 and 15) in whom this type of procedure was carried out presented extremely extensive disease involving the carina and right lateral tracheal wall, with the apparent site of origin of the bronchiogenic carcinoma in the right upper lobe orifice. At the time of surgery it was found necessary to remove extremely extensive portions of the tracheal wall, the carina, and one inch of the medial wall of the left main-stem bronchus. Attempts at repositioning of the trachea and left main-stem bronchus were not possible, so that it was necessary to construct an artificial trachea out of a previously constructed glass tube. This was placed with special attention not to allow the lower portion of the tube to occlude the left upper lobe orifice by its left lower lateral surface. This artificial trachea was then surrounded by a large pleural graft. The postoperative course of this patient did not present the difficulties associated with right lateral displacement of the mediastinum as had been noted with the reconstructed tracheobronchial junctures in which no foreign body was utilized. However, this patient in whom a glass tube had been utilized in order to reconstruct the trachea presented considerable difficulties with excessive secretions and it was extremely difficult to control the resultant pneumonitis in the left lung. Despite the presence of a large tracheotomy, it was difficult to maintain adequate aeration and a clear airway. The patient developed an empyema in the right pleural cavity. No evidence of tracheopleural or bronchopleural fistula was present. The patient died twenty days following operation and at autopsy small areas of chronic pneumonitis were found in the left lung, but felt not adequate in amount to explain the patient's death. The pleural graft was found to be intact, and the glass tube had maintained a good position. There was a definite superficial necrosis of the bronchial mucosa at all points in which the glass tube had been in contact. The empyema in the right pleural cavity appeared to be under control. No evidence of persistent car-

Certainly some patients in whom the disease is found at this stage may have asymptomatic extension of the neoplastic process to the liver or other silent areas. Also, there will be a definite percentage of patients presenting this syndrome in whom the lesion may prove inoperable at the time of exploration. Furthermore, it is hoped that with the increasingly early diagnosis of primary carcinoma of the lung, that extension upon the tracheal wall, or to the carina, will be less frequently seen. Certainly the procedure appears justified in the presence of a so-called bronchial adenoma, in which case it is potentially curative. Whether or not true primary carcinoma can be salvaged in this matter awaits further clinical observation and trial. In our opinion, definite palliative benefit is obtainable in this type of lesion.

The obstructive potential to the lumen of the airway, inherent upon the cartilage on the lateral aspect at the juncture of the trachea and left main-stem bronchus has been strongly demonstrated in our experience. It is hoped that experimental investigation will allow a satisfactory operative maneuver directly upon this cartilage to eradicate this complication. Until such time that this maneuver is obtained, extreme attention must be paid to maintaining the mediastinal contents slightly to the left of midline following this type of radical right pneumonectomy. Certainly it may be assumed, in view of the dramatic experimental work of Daniel relative to the construction of an artificial trachea, that the most satisfactory reconstruction of the tracheobronchial pathway should ultimately utilize his method.

CONCLUSIONS

1. A survey of the literature as well as the author's personal cases, still reveals that satisfactory early diagnosis of primary bronchiogenic carcinoma has not yet been obtained in most cases.

2. Primary bronchiogenic carcinoma is frequently found to arise in an upper lobe, as manifested by the finding of 51 primary upper lobe lesions in our personal group of 112 patients with this diagnosis.

3. It is felt that primary upper lobe carcinoma tends to present symptoms in a later stage of disease, due to a lesser inherent tendency to infection than that which occurs in lesions primary in the lower lobes.

4. Primary upper lobe tumors frequently achieve delayed final diagnosis, due to confusion of disease arising in this area with pulmonary tuberculosis.

5. A survey of our own personal cases and those at the Barnes Hospital, St. Louis, reveals a much higher percentage of lesions discovered in an inoperable stage when the tumor arises in an upper lobe. The most frequently inoperable lesion found is that arising in the right upper lobe. This is felt due to: (a) the immediate proximity of the superior vena cava and (b) the ease with which this lesion may involve the tracheal wall and carina. Diodrast outlining of the superior vena cava should be used prior to exploration of any right upper lobe tumor.

6. The author has presented a method which is felt applicable to neoplastic lesions arising in the right upper lobe area which seems to allow an extension of the indications for resectibility.

apparently well, without evidence of recurrence of disease eight and nine months following operation. The fourth patient died due to metastatic disease two and one-half months following surgery. The fifth patient, in whom a glass tube was used for tracheal reconstruction, died on the twentieth postoperative day. The sixth patient was still within the first month of the postoperative course. Therefore, at the time of this writing two of the six operated patients have died. It is felt that the patient in whom the primary disease was a bronchial adenoma in which malignant degeneration had occurred has probably the best chance for eradication of the disease. Both this patient and the one with associated megacolon have been able to resume their preoperative occupations and have existed considerably longer, with much greater comfort, than other patients whom we have seen with carcinomatous involvement of the trachea and carina.



A.

B.

Fig. 16 (J. E. W.).—A, X-ray studies before radical right pneumonectomy. Carcinoma presented extension into trachea and left main-stem bronchus. Massive defect following resection spanned by use of a glass tube and pleural graft.

B, X-ray studies after radical right pneumonectomy. Glass tube is visible on the post-operative film.

Despite the use of irradiation, palliative repeated bronchoscopic extirpation of obstructing tumor tissue and nitrogen-mustard therapy, the majority of patients whom we have seen with carcinoma involving the carina have died within two months following making of the diagnosis, with the longest survival time being six and three-fourths months.

Despite attempts at palliation by the usual conservative measures, patients with tracheal and/or carinal extensions of carcinoma rarely, if ever, regain a useful socio-economic status. In fact, they routinely follow a rapid downhill course of painful invalidism and progressive dyspnea. The rehabilitation seen in four of the six patients to date would appear to justify fully this procedure.

PHRENIC NERVE PARALYSIS

SPECIAL CONSIDERATION OF THE ACCESSORY PHRENIC NERVE

WINFIELD O. KELLEY, M.D., F.A.C.S.
NORWICH, CONN.

IT HAS seemed appropriate to make a careful study of the phrenic and accessory phrenic nerve relationships because of the fact that phrenic operations are employed quite extensively in treating certain types of pulmonary tuberculosis and because there is some lack of unanimity concerning the prevalence of accessory phrenic nerves.

Many authors have reported statistics on the incidence of accessory phrenic nerves found on cadaver dissections or at operation. Decker,¹ in dissections on seventy-two cadavers, found accessory nerves in 30 per cent, while at operation he was able to locate accessory phrenic nerves in only 15 per cent. Greenfield and Curtis² reported 24.4 per cent accessory nerves in 119 phrenic operations. Felix³ stated that the accessory phrenic nerve frequency was between 20 and 30 per cent. Sauerbruch⁴ found accessory nerves in 37.8 per cent. Ruhemann,⁵ in dissections of seventeen cadavers, found accessory phrenic nerves in more than 50 per cent. Fisher⁶ reported accessory nerves in 60 per cent of thirty-six operations. Aycock and Habliston⁷ found accessory nerves entering the main phrenic nerve below the usual site of operation in 65 per cent of 130 cadavers. Goetze,⁸ in dissections of twenty-five phrenic nerves on thirteen cadavers, found that phrenicotomy alone would have been unsuccessful in producing paralysis in 68 per cent. Alexander⁹ estimated that "one or more accessory roots (some of which were probably the nerve to the subclavius) have been found in 99 per cent of the initial phrenic operations" in his clinic.

In order to arrive at an accurate conclusion concerning the prevalence of accessory phrenic nerves, it has been necessary to decide upon certain criteria which could be applied to the cases studied. There is practically never any question concerning the identity of the main phrenic nerve. This can usually be confirmed by the production of a diaphragmatic movement upon stimulation. If two main phrenic trunks are found to course downward along the anterior scalene muscle, neither one of these is classified as an accessory nerve. When stimulation of another nerve produces a definite diaphragmatic response, this nerve is considered to be an accessory phrenic nerve. When the main phrenic nerve has been paralyzed at operation and the ipsilateral hemidiaphragm continues to move, it is assumed that an accessory nerve exists. If, after that nerve has been located and severed, the diaphragm ceases to move normally, that is also conclusive proof of the existence of an accessory phrenic nerve. These criteria were used in the collection of statistics in this paper.

Two series of consecutive cases were studied, one of which was collected in Saranac Lake, N. Y. Sept. 14, 1944 to April 18, 1946, and the other in the

From State Tuberculosis Sanatoria of Connecticut.

Read (by title) at the Twenty-eighth Annual Meeting of The American Association for Thoracic Surgery, Quebec, Canada, May 31, June 1, 2, 1948.

7. It is felt that this technique of resection should be utilized not only in patients in whom there is visible extension of tumor upon the tracheal wall and carina, but also in order to allow resection of an adequate margin of tissue proximal to carcinoma primary in the upper lobe bronchus. This should increase the potential cure rate.

8. The use of stabilization sutures has in our experience eradicated all of the dangers previously considered inherent upon the "open bronchial stump." With these sutures a defect 9 by 3 cm. in the lateral tracheobronchial wall has been left open without delivering oxygen or positive pressure to the remaining lung for a thirty-two minute interval without producing severe anoxia.

9. Experiences with the application of this procedure in six human patients are described, four in whom primary plastic reconstruction has been carried out. In two patients an artificial glass trachea was utilized. Certain complications attendant to this type of procedure are described in detail, as well as the methods utilized to counteract them.

10. It is felt that when no firm artificial support is utilized in this type of procedure, that constant attention must be given toward maintenance of the mediastinal structures slightly to the left of the midline. This may be maintained during the immediate postoperative period by the injection of air within the right pleural cavity, but soon requires a less compressible and more slowly absorbed substance such as plasma. The author has hesitated to utilize foreign bodies within the right pleural cavity such as Lucite balls, for fear of pressure erosion upon the suture line, or compression on the newly reconstructed airway.

11. It appears in patients in whom this procedure has been performed, that displacement of the mediastinum toward the right may produce acute or chronic gastric dilatation with pyloric obstruction. Correction of this complication has been achieved only by satisfactory reposition of the mediastinal contents. The mechanism of this reflex is not at present understood but does not appear to be produced by impulses carried by or produced through disturbance of the vagus nerves.

REFERENCES

1. Hopkins, W. A., and Abbott, O. A.: Modern Clues in the Diagnosis and Treatment of Primary Carcinoma of the Lung, *J. M. A. Georgia*, November, 1948.
2. Graham, E. A.: Personal data obtained from Surgical Chest Service of Barnes Hospital, St. Louis, Mo., 1945.
3. Griess, D. F., McDonald, J. R., and Clagett, O. T.: The Proximal Extension of Carcinoma of the Lung in the Bronchial Wall, *J. THORACIC SURG.* 14: 362, 1945.
4. Daniel, R. A.: The Regeneration of Defects of the Trachea and Bronchi, an Experimental Study, *J. THORACIC SURG.* 17: 335, 1948.
5. Beecher, H. K., and Linton, R. D.: Epinephrine in Cardiac Resuscitation, *J. A. M. A.* 135: 90, 1947.
6. Graham, E. A., and Bell, R. D.: Open Pneumothorax: Its Relation to the Treatment of Empyema, *Am. J. M. Sc.* 156: 839, 1918.

along the lateral border of the anterior scalene muscle, a portion of the brachial plexus can be exposed as it courses downward and somewhat laterally beneath the clavicle. The accessory phrenic nerve can be found as it leaves the fifth root of the plexus and proceeds downward and somewhat medially. This may occur at a rather low level so that it is sometimes necessary to retract the



Fig. 1.—Phrenic ring placed at the left upper corner of operating table for right phrenic operation. Ring measures 10.5 inches in diameter. Transverse bar extends 3 inches beyond ring, and vertical bar is 18 inches in length.



Fig. 2.—Patient and ring in place for right phrenic operation. Head is turned to left side and neck is supported by a small pillow.

brachial plexus by gently grasping its sheath with a fine hemostat and lifting upward (Fig. 5). The accessory phrenic nerve will be found often to branch in the form of a "Y." The lateral branch is the nerve to the subclavius muscle, and the medial branch is the typical accessory phrenic nerve which eventually joins the main phrenic trunk in the chest a few centimeters below the level of the clavicle. A portion of this branching nerve, for a distance of about 1 cm., is

Connecticut State Tuberculosis Sanatoria, July 25, 1946 to June 5, 1947. All patients had at least one phrenic operation, and several repeat paralyses were performed. Some patients had the primary operation performed by another surgeon. If an accessory phrenic nerve were found on the next operation (by the author), such cases were included in this series.

In the entire series of 309 cases, there was conclusive proof of the existence of at least one accessory phrenic nerve in 75.7 per cent. It should be noted that another nerve in addition to the main phrenic nerve was found in 99.2 per cent. There is a definite possibility that some accessory phrenic nerves are so small that the impulses carried by them to the diaphragm are too weak to be detected by the operator. It is conceivable that these nerves, if allowed to remain intact, could gain sufficient power to cause the diaphragm to function quite well after a period of a few weeks or months. Many times a branching nerve was located. If no diaphragmatic response was noted upon stimulation of this nerve, it was not classified as a definite accessory nerve. It seems fair, however, to call this presumptive evidence of an accessory phrenic nerve. There are many anatomic dissections^{1, 10} to show that the nerve to the subclavius muscle gives off a medial branch which joins the phrenic nerve. If these cases are included, the percentage increases from 75.7 to 80.9 per cent.

Because of the fact that there is occasionally some difficulty in locating the accessory phrenic nerve at operation, it seems appropriate to stress some specific points in technique which have proved to be of considerable practical importance. The patient is placed in the supine position with the head turned away from the side of operation. A small pad is placed under the back of the neck. It is most important to have a good view of the anatomic structures posterior to the clavicle. In order to obtain such a view, the operator must be able to stand at the upper end of the operating table and gaze downward into the depths of the incision. The regulation-type anesthesia screen, which is placed ordinarily over the patient's head before draping, is unsatisfactory, because it interferes with this view. A special piece of apparatus (Fig. 1) has been devised which holds the drapes away from the patient's head and also gives the surgeon adequate room in which to work. This consists of a metal ring held in a horizontal position just above the patient's head by an upright bar which is fastened to the corner of the operating table. The position of the drapes is illustrated in the accompanying photographs (Figs. 2, 3, and 4). The usual phrenic incision is made parallel to and about 2 cm. above the clavicle just lateral to the clavicular portion of the sternocleidomastoid muscle. After severing a few of the platysma muscle fibers, the sternocleidomastoid muscle is retracted medially to expose the layer of fatty tissue directly over the anterior scalene muscle. This tissue is separated easily, and the phrenic nerve is almost invariably located at this level just as it crosses the anterior surface of the anterior scalene muscle. The sheath (of prevertebral fascia) is incised for a very short distance so that the nerve can be isolated, infiltrated with Novocain, and crushed. It is advisable to explore the anterior and medial aspects of the anterior scalene muscle for a second phrenic trunk. The operator then shifts his position from the side to the head of the operating table. By blunt dissection

most of these instances, another small accessory phrenic nerve will be found extending medially from the brachial plexus at a very low level beneath the clavicle.

There is some lack of agreement concerning the proper method to be used in testing for and determination of complete paralysis of the hemidiaphragm. Some authors² (Greenfield and Curtis) stated that, "The best method which we have found for demonstrating a successful paralysis of the hemidiaphragm is the 'sniff test.'" Alexander³ stated that, "Hitzenberg's sniffing test is simple and especially useful." However, he continued, "When the patient sniffs suddenly and deeply a paralyzed, or even an incompletely paralyzed, hemidiaphragm is seen to undergo pronounced paradoxical movement." A positive "sniff test" indicates, therefore, that the affected leaf of the diaphragm is weaker than the opposite or normal side. Even though paradoxical motion is

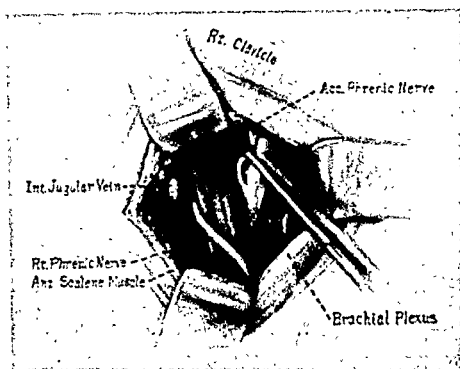


Fig. 5.—The right phrenic and accessory phrenic nerve viewed from above. The accessory nerve has been retracted upward by means of a hemostat. The medial branch extends downward beneath the clavicle to join the phrenic nerve, and the lateral branch is the nerve to the subclavius muscle.

noted on sniffing, another test is necessary to determine the presence or absence of complete paralysis. The most satisfactory test is performed as follows: The patient is asked to inhale deeply while the observer presses his two hands against the lower lateral portions of the ribs and watches the diaphragm fluoroscopically. A paralyzed hemidiaphragm will not move downward during this maneuver. If pressure is not exerted against the ribs, a costal breather may cause the ribs to flare sufficiently to produce a slight downward motion of a paralyzed diaphragm. If the diaphragm descends even slightly when the ribs are held in a fixed position, one should be strongly suspicious of a functioning accessory phrenic nerve in spite of the fact that there is paradoxical motion during the "sniff test." On several occasions, patients with these findings have been returned to the operating room and another accessory phrenic nerve has been located. Subsequent fluoroscopy almost invariably has shown complete paralysis of the hemidiaphragm with the deep-breathing, rib-holding test.

excised. Other accessory nerve fibers may be found at a higher or lower level. These are excised also so that it will not be necessary to explore this portion of the neck at a subsequent operation. A great variety of phrenic and accessory phrenic nerve relationships has been described by several authors. No attempt will be made to review this literature.^{1, 10, 11}

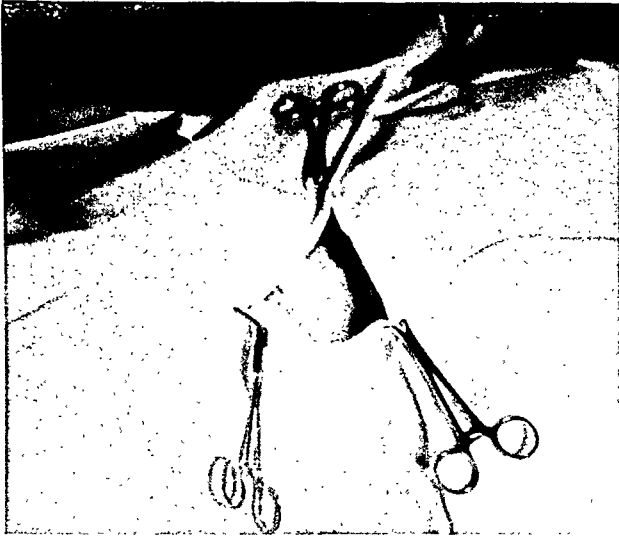


Fig. 3.—Drapes in place for right phrenic operation. The first three towels are placed to leave a triangular opening above the right clavicle. The fourth towel is draped across the ring over patient's head. This arrangement makes it possible for operator to stand above patient's head and look downward into incision.

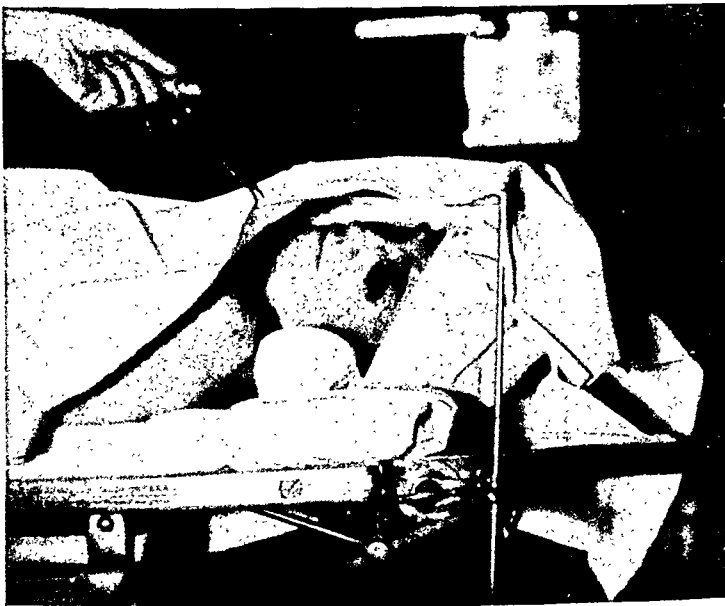


Fig. 4.—View from left side of table shows ample room for exchange of air.

After closure of the incision, the patient is taken immediately to the fluoroscopic room. If the leaf of the diaphragm on the side of operation is found to be moving, a re-exploration of the neck is carried out at once. In

ELECTROPHRENIC RESPIRATION

IV. THE EFFECTIVENESS OF CONTRALATERAL VENTILATION DURING ACTIVITY OF ONE PHRENIC NERVE

STANLEY J. SARNOFF, M.D., EDWARD A. GAENSLER, M.D.,* AND
JAMES V. MALONEY, JR., M.D.
BOSTON, MASS.

IT HAS been demonstrated that the application of a cyclically undulating electrical potential to one phrenic nerve produces effective artificial respiration in the dog, cat, rat, monkey, rabbit, and man. The arterial oxygen and carbon dioxide levels could be maintained within normal limits by this method.¹⁻⁴ Complete control of the rate and depth of respiration could be maintained since, during electrophrenic respiration, motor activity of the respiratory center is suppressed.^{3, 4} The mechanism of this respiratory suppression is largely vagal.

The fact that normal arterial oxygen levels could be maintained with the stimulation of only one phrenic nerve led to the assumption that one of two events was occurring in the contralateral lung during unilateral phrenic stimulation. Either the blood vessels of the contralateral lung, on which side the phrenic nerve was not being stimulated, were intensely constricted, thus shunting blood through the ipsilateral aerated lung, or, as seemed more likely, the contralateral lung was also being aerated. Jacobus⁵ technique of differential bronchspirometry, whereby the relative ventilation of the right and left lung could be accurately determined, offered the most direct means of determining how much contralateral aeration was occurring during unilateral phrenic stimulation.

In previous communications concerning electrophrenic respiration, only those experiments were described in which nerve stimulation was accomplished by direct contact between the electrode and nerve. More recently, it has been found possible to produce electrophrenic respiration in man by placing the stimulating electrode on the skin directly over the motor point of the phrenic nerve just as it crosses the lateral ridge of the scalenus anticus muscle. The characteristics and results of electrophrenic respiration produced by external stimulation are the same as those obtained with the direct contact technique.⁶

METHOD

After anesthetization of the tracheobronchial tree with 0.25 per cent pontocaine, a bronchspirometric catheter of the type devised by Zavod⁷ was introduced and the tip placed in the left main-stem bronchus under direct fluoroscopic control before inflating the retaining cuffs. The position of the bronchial catheter was checked by fluoroscopy and spot films during and after the procedure. The air spaces of the right and left lungs were then connected to

From the Department of Physiology, Harvard School of Public Health, and The Thoracic Surgery Service, Boston City Hospital.

Aided by a grant from The National Foundation for Infantile Paralysis.

Received for publication Sept. 16, 1949.

*Research Fellow, United States Public Health Service.

It is not the purpose of this paper to evaluate the benefits of phrenic nerve paralysis in pulmonary tuberculosis. Many authors^{9, 12, 13, 14} have shown statistically that certain varieties of the disease improve more rapidly with a paralyzed diaphragm so that the phrenic operation has been widely accepted as a helpful procedure.

CONCLUSIONS

1. The importance of finding the phrenic accessory nerve in order to produce complete paralysis of the hemidiaphragm is discussed.

2. A series of 309 cases is presented in which the incidence of accessory phrenic nerves (75.7 per cent) is greater than that reported by most authors.

3. Details of technique of the phrenic operation are discussed with emphasis on aids to facilitate the location of accessory phrenic nerves.

4. An apparatus to provide better exposure of the field of operation is described and illustrated.

5. An accurate method of testing for complete paralysis of the hemidiaphragm is described.

REFERENCES

1. Decker, H. R.: *J. THORACIC SURG.* 2: 543-548, 1933.
2. Greenfield, J., and Curtis, G. M.: *J. THORACIC SURG.* 12: 79-85, 1942.
3. Felix, W.: *Deutsche Ztschr. f. Chir.* 171: 283, 1922.
Idem: *Zentralbl. f. Chir* 49: 1832, 1922.
4. Sauerbruch, F.: *München med. Wehnschr.* 60: 625, 1913. *Ibid.* 70: 693, 1923.
5. Ruhemann, E.: *Beitr. z. Klin. d. Tuberk.* 59: 553, 1924.
Idem: *Arch. f. klin. Chir.* 139: 557, 1926.
6. Fisher, L.: *Am. Rev. Tuberc.* 25: 497, 1932.
7. Aycock, T. B., and Habliston, C. C.: *Am. Rev. Tuberc.* 22: 757, 1930.
8. Goetze, O.: *Arch. f. klin. Chir.* 121: 224, 1922. *Ibid.* 134: 594, 1925.
Idem: *München med. Wehnschr.* 72: 1110, 1925.
9. Alexander, John: *The Collapse Therapy for Pulmonary Tuberculosis*, Springfield, 1937, Charles C Thomas, Publisher, pp. 133, 137, 190-193.
10. Yano, K.: *Folia Anatomica Japonica* 3: 95-106, 1925.
11. Plenck, A., and Matson, R. C.: *Beitr. z. Klin. d. Tuberk.* 62: 350-355, 1926.
12. Nehil, L. W., and Alexander, John: *J. THORACIC SURG.* 2: 559-569, 1933.
13. O'Brien, E. J.: *J. A. M. A.* 95: 650, 1930.
14. Wallaston, F. L.: *Tubercle* 24: 121-123, 1943.

performed during electrophrenic respiration demonstrated that this sequence of diaphragmatic and mediastinal motions observed in Fig. 1 occurred under the experimental conditions described previously.

Differential bronchspirometry tracings were obtained twenty-one times during electrophrenic respiration and spontaneous respiration in seven subjects. The tracings were all suitable for the determination of differential ventilation. Nine tracings were taken for a sufficient length of time to be adequate for determinations of oxygen consumption.

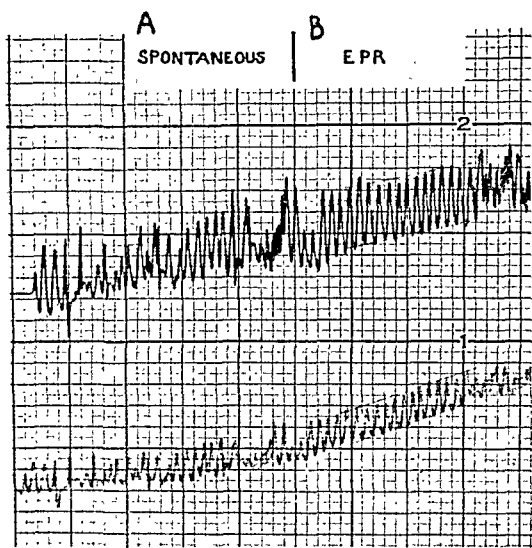


Fig. 2.—Differential bronchspirometry during spontaneous and right electrophrenic respiration. A, spontaneous respiration; B, electrophrenic respiration. Upper tracing, right lung; lower tracing, left lung (see Table I).

The results are listed in Table I. During electrophrenic respiration by means of unilateral phrenic stimulation, the contralateral lung was always adequately ventilated. At a rate close to that of the patient's spontaneous rate, electrophrenic respiration produced relative ventilation of both lungs not significantly different from the relative ventilation observed during spontaneous respiration (Patients J. P. and W. R.). As the rate was increased, the proportion of the total ventilation performed by the contralateral lung sometimes decreased (Patients J. P., W. R., J. M., and M. M.). The magnitude of this change, however, was never striking. The decrease in percentage ventilation of the contralateral lung never exceeded 12 per cent of the total at electrophrenic rates up to twice the spontaneous rate. This was true despite the fact that, in

a Gebauer twin spirometer which recorded on moving paper. In subsequent illustrations, the upper tracing represents the ventilation of the right lung, the lower tracing that of the left lung.

Four patients with minimal tuberculosis, two with moderately advanced disease, and one normal volunteer were studied by differential bronchspirometry in respect to a comparison of electrophrenic respiration and spontaneous respiration. These patients were found to have normal or almost normal combined ventilatory function as established by maximal breathing capacity, vital capacity, walking ventilation, and spirometry. After several minutes of spontaneous respiration, during which time the rate, differential tidal volumes, and oxygen uptake of the two lungs were obtained, electrophrenic respiration was started. Tidal volumes, rate, and oxygen uptake from the two lungs were thus recorded during electrophrenic respiration and comparison made with the spontaneous values. Direct observation and, from time to time, fluoroscopic observation were used to be certain of the absence of spontaneous respiratory effort.

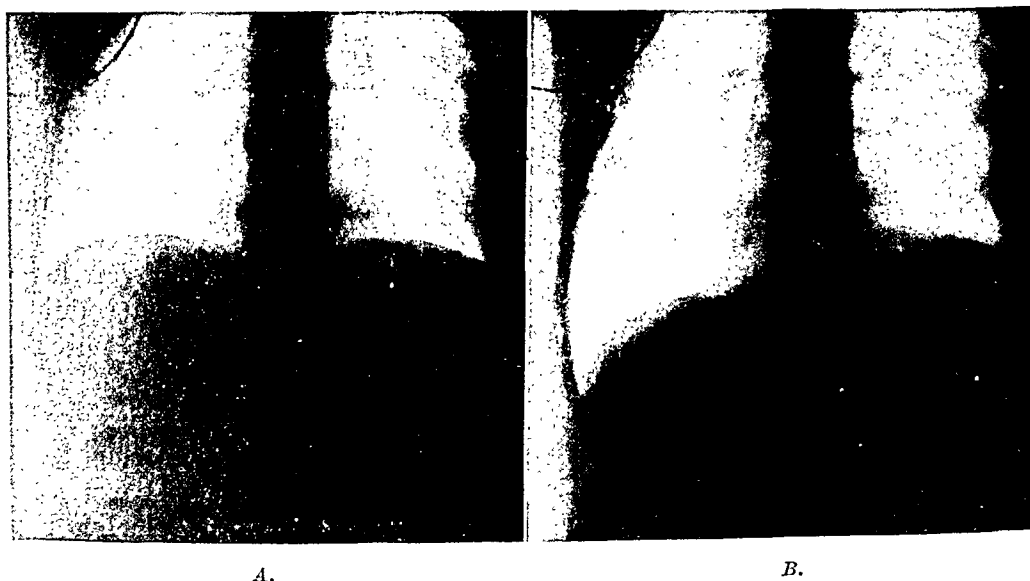


Fig. 1.—Roentgenogram of chest during electrophrenic respiration with stimulation of the right phrenic nerve: *A*, during the resting phase of expiration; *B*, at the peak of inspiration. Note the pronounced descent of the right diaphragm, slight elevation of the left diaphragm, and shift of the mediastinum to the right.

Serial chest films were taken in rapid succession on one of us (S. J. S.) during several full cycles of electrophrenic respiration in order to demonstrate the changes in the position of the right and left sides of the diaphragm as well as the mediastinum during electrophrenic respiration (Fig. 1).

RESULTS

Fig. 1 illustrates the effect of right-sided electrophrenic respiration on the position of the right and left sides of the diaphragm and on the mediastinum. During inspiration the right side of the diaphragm descends, the mediastinum shifts to the right, and the left side of the diaphragm is slightly elevated. The two roentgenograms shown in Fig. 1 were made in rapid succession. Fluoroscopy

every case, the minute ventilation during electrophrenic respiration was appreciably in excess of that during spontaneous respiration. In no case did the percentage decrease of contralateral lung ventilation diminish more than 16 per cent of the total, even at exceedingly rapid rates.

Figs. 2 and 3 show recordings of differential ventilation obtained during spontaneous and then electrophrenic respiration.

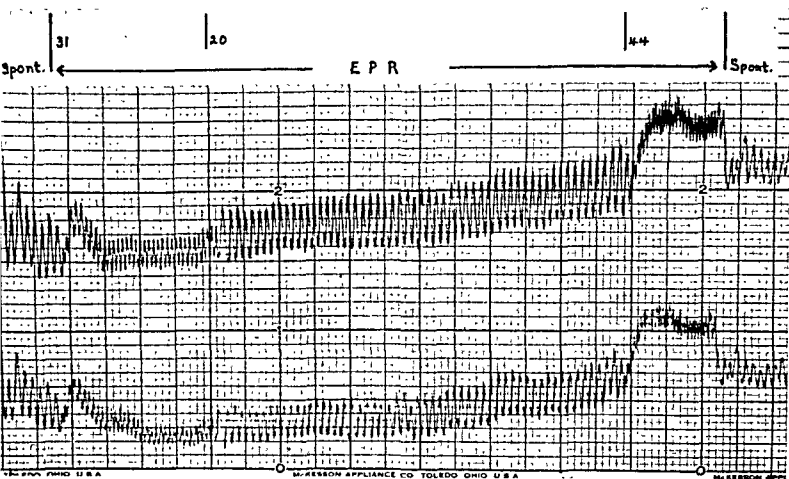


Fig. 3.—Differential bronchspirometry during spontaneous and right electrophrenic respiration at varying rates. Patient W. R. in Table I. Spontaneous respiration followed by EPR at rate of 31 per minute, 20 per minute, and 44 per minute. The last segment is again spontaneous respiration. Note the similarity of the differential ventilation during spontaneous and electrophrenic respiration.

DISCUSSION

The preceding data resolve the question of the mechanism whereby unilateral stimulation results in normal arterial oxygen saturation during electrophrenic respiration. It is clear that bilateral pulmonary ventilation occurs. It has previously been demonstrated that, within a few seconds after the onset of electrophrenic respiration, spontaneous respiratory effort ceases.^{3, 4} In addition to published data on this point, data are available from animal experiments in which action potentials were recorded from the unstimulated side of the diaphragm and intercostal muscles before and during right-sided electrophrenic respiration. They showed that all respiratory motor impulses ceased during electrophrenic respiration.⁸ This being the case, the ventilation of the contralateral lung must, in some way, be related to the motion of the ipsilateral (stimulated) diaphragm. The stimulated side of the diaphragm descends and expands the lung on that side; the mediastinum shifts toward the stimulated side in response to differentially created negative intrapleural pressure, thus expanding

TABLE I. DIFFERENTIAL VENTILATION, OXYGEN CONSUMPTION, MINUTE VOLUMES, AND VENTILATORY EQUIVALENTS DURING SPONTANEOUS AND UNILATERAL (RIGHT) ELECTROPHRENIC RESPIRATION

PATIENT	TYPE OF RESPIRATION	RATE	VENTILATION				O ₂ CONSUMPTION				MINUTE VOLUME (C. C.)	VENTILATORY EQUIVALENT		REMARKS
			PER CENT RIGHT	PER CENT LEFT	PER CENT RIGHT	PER CENT LEFT	PER CENT RIGHT	PER CENT LEFT	PER CENT RIGHT	PER CENT LEFT		RIGHT	LEFT	
A. R.	Spontaneous EPR	12	53	47	52	48	—	—	—	—	6,960	2.2	2.1	Normal volunteer
		48	53	47	—	—	—	—	—	—	7,220	—	—	—
M. M.	Spontaneous EPR	15	51	49	37	63	37	63	3.9	2.2	6,750	3.9	2.2	Pulmonary tuberculosis, moderately advanced,
		24	63	37	35	65	35	65	6.9	2.2	10,800	6.9	2.2	asymptomatic; small
		25	61	39	36	64	36	64	5.0	1.7	10,370	5.0	1.7	area of cavitation, right
		48	67	33	—	—	—	—	4.5	2.4	7,520	4.5	2.4	upper lobe
J. P.	Spontaneous EPR	10	64	36	63	37	63	37	2.2	2.1	5,150	2.2	2.1	Pulmonary tuberculosis, minimal; infiltrative disease, left upper lobe
		12	64	36	—	—	—	—	—	—	5,280	—	—	—
		18	72	28	67	33	67	33	2.5	2.0	7,200	2.5	2.0	—
		22	75	25	—	—	—	—	—	—	5,520	—	—	—
		34	76	24	—	—	—	—	—	—	9,860	—	—	—
		18	56	44	60	40	60	40	3.3	4.0	8,550	3.3	4.0	Pulmonary tuberculosis, minimal, asymptomatic; minute infiltrative lesion, left upper lobe
W. R.	Spontaneous EPR	20	58	42	60	40	60	40	6.0	6.3	12,000	6.0	6.3	—
		31	63	37	—	—	—	—	—	—	11,010	—	—	—
		31	65	35	—	—	—	—	—	—	10,550	—	—	—
		42	67	33	59	41	59	41	3.9	2.7	13,870	3.9	2.7	—
		44	58	42	—	—	—	—	—	—	13,640	—	—	—
R. P.	Spontaneous EPR	15	60	40	67	33	67	33	1.9	2.5	5,480	1.9	2.5	Pulmonary tuberculosis, minimal; left apical infiltration, left pleural effusion
		27	69	31	67	33	67	33	2.2	2.1	7,510	2.2	2.1	—
P. F.	Control EPR	9	58	42	18	82	18	82	8.8	1.3	9,000	8.8	1.3	Pulmonary tuberculosis, moderately advanced; multiple cavitation, upper half right lung
		25	60	40	21	79	21	79	9.4	1.7	15,620	9.4	1.7	—
J. M.	Spontaneous EPR	12	67	33	60	40	60	40	2.6	1.9	6,600	2.6	1.9	Pulmonary tuberculosis, minimal, asymptomatic; minimal bilateral, apical infiltration
		18	75	25	—	—	—	—	—	—	10,090	—	—	—
		22	75	25	61	39	61	39	2.3	1.2	10,560	2.3	1.2	—
		24	77	23	61	39	61	39	2.3	1.1	10,320	2.3	1.1	—
		32	80	20	—	—	—	—	—	—	11,800	—	—	—
		32	80	20	—	—	—	—	—	—	16,000	—	—	—
		36	83	17	—	—	—	—	—	—	15,120	—	—	—

2. The mechanism which we hold responsible for the ventilation of the lung on the nonstimulated side is mediastinal shift. This can be demonstrated by fluoroscopy or serial roentgenograms with and without the obstructing catheter in situ (Fig. 1).

3. The rise of the functional residual air from the resting base line (or midcapacity) which occurs when one diaphragm is stimulated at a much higher than resting rate occurs on both sides to an equal or almost equal extent. This illustrates not only that stenosis effects do not greatly, if at all, favor the stimulated side, but also that the force which causes an inrush of air into the lungs is equally applied to both sides, or almost so (Fig. 3).

Phrenic Nerve Interruption.—The fact that the mediastinum can swing sufficiently to maintain normal ventilation of the lung opposite to the stimulated side is of great interest since electrophrenic stimulation is used as a means of artificial respiration. It is further of interest in connection with the interruption of the phrenic nerve to provide pulmonary collapse and "rest" in the treatment of tuberculosis.

In order to re-evaluate any benefit of such an operation in the light of the above data, it is first necessary to attempt to define pulmonary collapse and "rest." Because of the ever-recurring confusion in the literature, it must be pointed out that collapse and rest are not synonymous. Collapse therapy is usually defined as any procedure which will cause a reduction in lung volume. "Rest" implies "functional elimination" or reduction in the share of work, for example, ventilation which the lung is called upon to perform. Which of these factors play a dominant role in the therapeutic effect varies with the type of treatment. Thus the often dramatic improvement which follows a period of bed rest is due, only to some extent, to collapse following the reduction in lung volume incident to the horizontal position of the body. Rest here appears to play a major role. In certain other situations rest enters the picture only to a very small extent, if at all. Pneumothorax induced on an only remaining lung following contralateral pneumonectomy or pneumoperitoneum with equal bilateral rise of the diaphragms are cases in point.

There appears to be no question that unilateral phrenic nerve interruption causes a degree of collapse or reduction in lung volume. Measurement on corpses has shown a decrease of 15 to 30 per cent of the volume of the involved hemithorax following phrenic nerve interruption.⁹ Lindskog and Friedman¹⁰ studied lung volumes in seven cases before and after temporary phrenic nerve interruption. They were able to show an average reduction of about 10 per cent of the functional residual air after operation. There appears to be less agreement of the effect of diaphragmatic paralysis on vital capacity and maximum breathing capacity. While Gaubatz¹¹ and Rutgers and Smilovits¹² reported reduction in these values of as much as 30 per cent, Warring,¹³ Wright,¹⁴ and Wright and Woodruff¹⁵ found little or no alterations in maximum breathing capacity and vital capacity following this operation. In nine patients studied before and fourteen days after phrenic nerve interruption we found a mean reduction of 10 per cent of maximum breathing capacity and 16 per cent of vital capacity.¹⁶ Two variables make comparison of such studies difficult. The first

the contralateral lung, almost to the same degree as the lung on the stimulated side. The uncontracted contralateral leaf of the diaphragm is elevated slightly, its ascent probably being limited in part by its continuity with the contracted side of the diaphragm through the central tendon.

Tracings in Figs. 2 and 3 and the data in Table I demonstrate that the differential tidal volume percentages and oxygen consumption of the lung on the unstimulated side during electrophrenic respiration approach the values obtained during spontaneous respiration.

Stenosis Breathing.—Because of the very special circumstances surrounding the bronchspirometric technique, conclusions arrived at by this method are limited in two respects. First, the patient is not under basal conditions and thus successive data obtained are not always comparable as to their absolute values. The relative percentage which each lung contributes to the total performance does, however, remain the same in the absence of unilateral interference with pulmonary function. Any change or lack of change in these relative percentages is thus significant. With the exception of minute ventilation, only such relative data have been reported herein and conclusions drawn pertain only to them. Second, because the bronchspirometric catheter has to contain within its envelope four separate lumina—two for the airways and two for the inflatable cuffs—and still must be small enough to pass into the trachea without injury to the vocal cords, a certain amount of stenosis breathing cannot be avoided. This effect is small or negligible at resting minute volumes but becomes so large at high air flow velocities that ratios of maximal ventilatory efforts obtained by this method are of little value. The present data might thus be questioned in that electrophrenic stimulation may create greater than resting air flow velocities. However, by this method, diaphragmatic motion can be controlled both in extent and in rate to such a degree that the spontaneous resting minute volume and resting respiratory rate can be closely simulated (Table I and Fig. 2). Several of our tracings illustrate the fact that only when the diaphragm is stimulated to exchange much larger than spontaneous volumes of air at several times the resting rate does the effect of stenosis breathing become apparent in some instances. The passive contractile force of the lungs and thorax is then unable to expel the entire volume of air which was forcibly introduced during inspiration by the powerful action of the diaphragm. The functional residual air thus increases but upon return to electrically controlled slower rates or spontaneous respiration it promptly returns to its former value (Fig. 3). Additional evidence can be presented that stenosis breathing effects do not invalidate results reported at or near resting respiratory rates and resting minute volumes:

1. In all cases presented the percentage of minute volume which each lung contributes with unilateral electrophrenic stimulation is equal or nearly equal to the spontaneous ratio at resting or near resting minute volumes. Only when the rate and minute volumes are much larger than the spontaneous one does the ratio of ventilation shift somewhat in favor of the stimulated side. It is exactly under these conditions that stenosis breathing begins to play a large role.

During this procedure it was found that ventilation of the contralateral as well as the ipsilateral lung occurred as determined by differential bronchspirometry.

2. This is caused by the shift of the mediastinum to the stimulated side thus causing the expansion of the contralateral lung. This is followed by a slight elevation of the unstimulated side of the diaphragm.

3. These data suggest that the therapeutic benefit from phrenic nerve interruption, if any, results primarily from partial parenchymal collapse rather than from decreased ventilation (rest).

REFERENCES

1. Sarnoff, S. J., Hardenbergh, E., and Whittenberger, J. L.: Electrophrenic Respiration, *Science* 108: 482, 1948.
2. Sarnoff, S. J., Hardenbergh, E., and Whittenberger, J. L.: Electrophrenic Respiration, *Am. J. Physiol.* 155: 1, 1948.
3. Whittenberger, J. L., Sarnoff, S. J., and Hardenbergh, E.: Electrophrenic Respiration. II. Its Use in Man, *J. Clin. Investigation* 28: 124, 1949.
4. Sarnoff, S. J., Whittenberger, J. L., and Hardenbergh, E.: Electrophrenic Respiration. III. Mechanism of the Inhibition of Spontaneous Respiration, *Am. J. Physiol.* 155: 203, 1948.
5. Jacobus, H. C.: Bronchspirometry: Review of Present Experiences and Some Further Investigations, *J. THORACIC SURG.* 7: 235, 1938.
6. Sarnoff, S. J., Maloney, J. V., and Whittenberger, J. L.: Electrophrenic Respiration. V. Effect on the Circulation of Electrophrenic Respiration and Positive Pressure Breathing During the Respiratory Paralysis of High Spinal Anesthesia. *Ann. Surg.* (In press.)
7. Zavod, W. A.: Bronchspirometry; Description of Catheter and Technique of Intubation, *J. THORACIC SURG.* 10: 27, 1940.
8. Chatfield, P. O., and Sarnoff, S. J.: The Role of Pulmonary Proprioception in the Suppression of the Respiratory Center During Electrophrenic Respiration. (In preparation.)
9. Brunner, A.: Die chirurgische Behandlung der Lungentuberkulose, Leipzig, 1924, Johann Ambrosius Barth.
10. Lindskog, G. E., and Friedman, I.: The Effect of Thoracoplasty and Phrenic Paralysis on the Total Volume of the Lung and Its Component Parts, *Am. Rev. Tuberc.* 34: 505, 1936.
11. Gaubatz, E.: Unbefriedigendes und weitere Entwicklungsmöglichkeiten in der operativen Behandlung der Tuberkulose, *Der öffentliche Gesundheitsdienst* 4: 361, 1938.
12. Ruttgers, P., and Smilovits, N.: Ueber roentgenologische und funktionelle Untersuchungsmethoden und ihre praktische Bedeutung für die Phthisiologie, *Helvet. med. acta.* 5: 474, 1938.
13. Warring, F. C., Jr.: Ventilatory Function: Experiences With Simple Practical Procedure for Its Evaluation in Patients With Pulmonary Tuberculosis, *Am. Rev. Tuberc.* 51: 432, 1945.
14. Wright, G. W.: Personal communications.
15. Wright, G. W., and Woodruff, W. W.: The Effect of Surgical Collapse Therapy on Pulmonary Function, Read Before the Medical Section of the American Trudeau Society at Chicago, May 11, 1944.
16. Guensler, E. A., and Strieder, J. W.: Pulmonary Function Before and After Extra-pleural Pneumothorax, *J. THORACIC SURG.* (To be published.)
17. Pinner, M.: Physiological Principles of Collapse Therapy, in Alexander, John: The Collapse Therapy of Pulmonary Tuberculosis, Baltimore, 1937, Charles C Thomas, Publisher, Chap. 3, p. 65.

is the extent of denervation of the diaphragm since accessory nerves are occasionally overlooked even if they are diligently sought. The second is the time elapsed between nerve interruption and ventilatory tests. Studies of others and our own are in agreement that any reduction in pulmonary volumes and breathing capacities, if they occur, return to the preoperative values long before diaphragmatic motion returns. A far better evaluation of reduction in lung volume might be obtained by the bronchspirometric technique employed before and after operation. No such reports have appeared in the literature. The well-known difficulties of reliable residual air determinations together with the problems involved in bronchspirometry may account for this. We have been successful in this attempt in only two cases. In both there was a satisfactory rise in the diaphragm in the treatment of minimal disease. In one, the lung volume on the phrenicectomized side was decreased 17 per cent four weeks after operation, and in the other, 11 per cent two weeks after operation.¹⁶

No factual evidence has been reported in the literature concerning the degree of rest which diaphragmatic paralysis affords. Even co-authors of the same book disagree on this point. While Pinner¹⁷ felt that the factor of rest is slight because of compensatory increase in lateral expansion of the chest and increased motion of the upper ribs, Alexander¹⁷ was "strongly of the opinion that paralysis of a freely moving diaphragm greatly rests the lung." In evidence he cited cases where clinical improvement was evident following phrenic paralysis in the absence of elevation of the diaphragm. The only adequate way to demonstrate a relative decrease in ventilation and blood flow of the lung on the denervated side is bronchspirometric evaluation. In nine cases studied by us and cited previously, the relative ventilation changed only 3 per cent after operation.¹⁶ In the absence of hypoxia it can also be presumed that there was no change in relative blood flow.

The present studies with electrophrenic respiration not only support the preceding findings but also offer an explanation for them, namely compensatory ventilation by mediastinal swing. At respiratory volumes and rates approximating those of the resting state, the mediastinum has been shown to provide a normal relative ventilation and oxygen consumption for the lung in the absence not only of diaphragmatic action but also of intercostal activity. Phrenic interruption may cause a certain amount of collapse or reduction in lung volume thus relaxing elastic tissues, promoting scar formation, and perhaps causing small local areas to rest due to differential elasticity of normal and diseased areas. On the other hand, in the absence of a fixed mediastinum, phrenic nerve interruption does not provide any degree of general rest for the lung because of the ventilatory effects of the mediastinal shift under these circumstances. The element of pulmonary rest as defined previously is, therefore, not likely to be an important factor in whatever therapeutic benefit the patient obtains from phrenicectomy.

CONCLUSIONS

1. Seven patients were respired by means of externally applied electrophrenic respiration capable of maintaining adequate arterial oxygen saturation.

right pneumothorax was attempted unsuccessfully. A right phrenic crush was done in 1937, which controlled the disease until 1942, when pneumoperitoneum was instituted for reactivation of disease. Between 1942 and 1947, when she was admitted to this hospital, repeated hemoptyses were noted and the lesion on the right had progressed to frank cavitation. The pneumothorax on the left was still present. Pulmonary function studies at that time revealed an M.B.C. of only 30.7 liters per minute and a B.R. of 23.5 liters per minute. All other studies (Table I) were likewise markedly reduced. It was felt that this patient's function could not support surgery at this time.

A program of graded activity was devised for this patient in an attempt to increase her ventilatory capacity. This consisted of breathing and walking exercises. Under this program the left lung re-expanded and when retested six months after admission the M.B.C. had risen to 36 liters per minute with an increase in B.R. to 29.4 liters per minute. Bronchspirometry showed the right lung to be carrying on 76 per cent of total oxygen consumption and 78 per cent of total ventilation. Further exercise enabled this patient to increase the M.B.C. to 56.7 liters per minute and B.R. to 49 liters per minute. Bronchspirometry showed the right lung to be carrying on 87 per cent of total oxygen consumption and 85 per cent of total ventilation respectively. At this time it was agreed to collapse the right apex by extrapleural Lucite plombage.

In June, 1947, the extrapleural pneumonolysis was performed. Postoperative course and convalescence were uneventful. Four months following surgery the patient was retested and showed an M.B.C. of 50 liters per minute, a loss of only 6 liters from the preoperative figure. B.R. was 44.2 liters, a loss of 4.86 liters per minute. Bronchspirometry showed the right lung to have 87 per cent of total ventilation and 75 per cent of total oxygen consumption.

Comment.—In this patient the problem was one of enabling her to reach a satisfactory total pulmonary function level before surgery could be attempted. Bronchspirometry then revealed that most of the function was in the right lung which was the lung to be collapsed. The extrapleural procedure was chosen because it was felt that the collapse could be confined to the involved portion of the lung and that encroachment on healthy respiring tissue could be reduced to a minimum. These views seem to be substantiated by postoperative study in which the total loss in the M.B.C. was 6 liters per minute.

B. M., a 37-year-old white woman, had tuberculosis dating from 1931. In 1933, bilateral upper lobe cavitation was present and the patient spent one year on bed rest. In 1934, attempted bilateral pneumothorax was unsuccessful. A left phrenic crush was done at this time. In 1935, right phrenic crush was performed. The patient's status had improved, sputum was negative, but the patient was still on bed rest. By 1940, the sputum was positive again, and the right phrenic was recrushed. The sputum remained positive following this latter procedure and continued consistently positive until 1948, when an extrapleural pneumonolysis with Lucite plombage was performed on the left.

Studies before surgery revealed a borderline function, with an M.B.C. of 40 liters per minute. Performance during exercise revealed mild dyspnea on the step test but recovery was rapid. Brouchspirometric evaluation showed the right lung to be carrying 77 per cent and 76 per cent of the total oxygen consumption and ventilation, respectively. Function testing was repeated three months after surgery, showing no decrease in total function in that the M.B.C. and performance on the step test was the same as in the preoperative period. Bronchspirometry now showed the right side to be carrying 73 per cent and 70 per cent of the total oxygen consumption and ventilation.

Comment.—This patient has the characteristic bilateral fibroid type of tuberculosis. Multiple phrenic operations plus obliteration of the pleural space had reduced the pulmonary function to the point where very little reserve was

PULMONARY FUNCTION STUDIES BEFORE AND AFTER EXTRAPLEURAL PNEUMONOLYSIS WITH PLOMBAGE

SIDNEY H. DRESSLER, M.D.,* GERALD J. BRONFIN, M.D.,† AND
JOHN B. GROW, M.D.‡
DENVER, COLO.

PRESERVATION of the maximal amount of pulmonary function compatible with adequate control of disease has always been the goal of collapse therapy in pulmonary tuberculosis. Development of newer methods in the evaluation of pulmonary function has made it possible to choose procedures for patients with markedly impaired function which will contribute no noteworthy reduction of function yet will adequately control the disease process.

For the past two years extrapleural pneumonolysis with Lucite (methyl methacrylate) plombage has been performed at this institution. A major indication for the use of this procedure has been in patients with such severely reduced pulmonary function that thoracoplasty, which would ordinarily be the method of choice, could not be done with reasonable assurance of postoperative survival. In others the extrapleural procedure was chosen because of the expectation of bilateral collapse therapy where the necessity for preservation of function is obvious.

This paper presents the results of function studies on nine patients before and after insertion of Lucite spheres in the extrapleural space. The methods of function testing have been adequately described elsewhere and need no elaboration here other than a brief statement of the various procedures used. These included: maximum breathing capacity (M.B.C.), breathing reserve (B.R.) from rest to voluntary maximum ventilatory effort, lung subdivisions, and oxygen consumption, plus ventilation in the recovery period after a standard exercise test. After completion of these studies from which an estimation of total pulmonary function was gained, divided function of the separate lungs was done using the bronchspirometric methods of Jacobaeus,¹ Gebauer,² and Zavod.³ It is from this latter procedure that the most illuminating information could be secured after it had once been shown that total function could sustain a surgical intervention.

CASE PRESENTATIONS

A. W., a 43-year-old white woman, had the onset of pulmonary tuberculosis in 1933, with a minimal lesion in the left apex. A left pneumothorax was initiated in 1933, and maintained for fourteen years. In 1935, a lesion was noted in the right apex for which

From the National Jewish Hospital at Denver, Colo., Allan Hurst, M.D., Medical Director, and the University of Colorado Medical Center.

Received for publication Sept. 30, 1949.

*Assistant Medical Director, National Jewish Hospital.

†Research Fellow in Cardio-Pulmonary Physiology; National Life Insurance Medical Research Fund.

‡Chief of Thoracic Surgery, National Jewish Hospital.

left. It was felt that an extensive surgical procedure involving collapse of respiring tissue would have in all probability relegated this individual to the ranks of the bedfast. The extrapleural plombage was performed as a calculated risk and it appeared not to have affected the patient's function to any appreciable extent.

C. B., a 29-year-old white woman had the onset of tuberculosis some fifteen years before admission to this institution. At different times she had had bilateral upper lobe cavitation for which she had received artificial pneumothorax and phrenic crush, both on the left. Artificial pneumothorax had been initiated some four years before admission to this hospital. On examination here the patient revealed an active process in the right upper lobe with cavitation and a paralyzed right hemidiaphragm. A therapeutic pneumothorax was present on the left. Sputum was highly positive. The pneumothorax on the left was re-expanded and pulmonary function testing was done. Total function was seriously diminished, the M.B.C. being 33.2 liters per minute, and vital capacity 1,150 c.c. The standard exercise test was performed with marked dyspnea and prolonged recovery. A program of breathing exercises and walking activity was started with the hope of increasing function. In six months the M.B.C. had risen to 38.6 liters per minute with definite improvement in the performance of exercise. Bronchspirometry showed the left lung to be carrying 89 per cent of the total oxygen consumption and 71 per cent of the ventilatory function. An extrapleural pneumonolysis with Lucite plombage was performed on the right. Retesting six months after surgery revealed some decrease in total pulmonary function with an M.B.C. of 32.0 liters per minute although there was no clinical evidence of increased dyspnea. Bronchspirometry showed the left lung to be carrying 95 per cent of total oxygen consumption and 93 per cent of total ventilation.

Comment.—This girl had an even more seriously diminished function than the previous case. Because of her youth and the weak prospects for recovery without surgical intervention, the plombage was performed. As demonstrated by the function testing, a decrease in total function did occur, but not of sufficient magnitude to incapacitate markedly this patient.

L. H., had initial tuberculosis, consisting of bilateral apical involvement with cavitation on the left, which was discovered some ten years prior to admission to this hospital. A left pneumothorax and left phrenicectomy failed to control this lesion, the right apex having become stabilized in the interim. X-ray examination at the time of admission showed fibrovascular disease in the left upper lung with a raised, paralyzed diaphragm on the left. The right lung showed apical fibrosis with some basal emphysema and upward retraction of the right hilar structures. Function studies showed an M.B.C. of 48.2 liters per minute with mild dyspnea on the standard exercise test. Bronchspirometry showed the right lung to be carrying 62 per cent of the total oxygen consumption and 70 per cent of the ventilation. Left extrapleural pneumonolysis with Lucite plombage was done. Retesting some four months postoperatively revealed a reduction in M.B.C. to 46.4 liters per minute, a loss of 1.8 liters with no change in the performance of exercise. Bronchspirometry showed the right lung to be carrying 66 per cent of total oxygen consumption and 72 per cent of total ventilation.

Comment.—This case demonstrates a minimal loss in function following the surgical collapse procedure. This negligible decrease may be interpreted as being due to collapse only of nonfunctioning parenchyma or, as demonstrated by Björkman⁴ and others, may possibly be a compensatory increase in the function of the contralateral lung.

The other five cases in this series, which will not be described in detail, are of the same order, in that loss in total function was held to a minimum. In one

TABLE I

BRONCHIOSPIROMETRY												
TOTAL FUNCTION											VITAL CAPACITY	
CASE	STATE	R.M.V. (LITERS PER MIN.)	V.C. (C.C.)	M.B.C. (LITERS PER MIN.)	B.R. (LITERS PER MIN.)	B.R. (%) AT REST	B.R. (%) 1ST MIN. REC.)	V.E.	SIDE (LUNG)	OXYGEN CONSUMPTION		
										C.C. PER CENT		
1. O. E., left ex- trapleural	Preop.	10.4	2,375	44.1	33.7	76	65	2.2	Right	250 125	975 625	61 39
	Postop.	10.0	2,175	43.0	33.0	76	65	1.4	Right Left	320 200	1,360 360	79 21
2. C. B., right extrapleural	Preop.	7.7	1,075	38.6	30.9	80	71	2.2	Right Left	375 88	175 425	29 71
	Postop.	6.8	1,100	32.0	25.2	79	61	2.1	Right Left	0 250	100 1,000	9 91
3. L. B., right extrapleural	Preop.	5.0	1,725	76.0	71.0	93	88	1.5	Right Left	50 325	275 1,375	17 83
	Postop.	4.2	1,485	75.0	70.8	94	89	1.2	Right Left	60 300	200 1,200	14 86
4. B. M., left extrapleural	Preop.	4.9	1,475	40.0	35.1	87	78	1.6	Right Left	250 75	1,010 325	76 24
	Postop.	6.0	1,270	41.0	35.0	85	79	1.8	Right Left	245 50	1,000 250	80 20
5. M. M., right extrapleural	Preop.	6.9	2,000	84.3	77.4	91	89	2.0	Right Left	210 210	1,000 500	67 33
	Postop.	7.5	1,800	98.0	90.5	92	90	2.3	Right Left	150 250	750 975	43 57
6. L. H., left extrapleural	Preop.	6.9	1,475	48.2	41.3	85	75	2.7	Right Left	200 125	1,075 450	70 30
	Postop.	6.8	1,425	46.4	39.2	85	72	2.7	Right Left	200 100	1,050 400	72 28
7. A. W., left extrapleural	Preop.	7.7	1,450	56.7	49.0	86	79	2.2	Right Left	325 50	1,075 190	85 15
	Postop.	5.8	1,200	50.0	44.2	88	80	1.6	Right Left	225 75	975 150	87 13
8. O. M., left extrapleural	Preop.	6.7	2,425	59.2	52.5	88	70	2.1	Right Left	525 0	450 1,400	25 75
	Postop.	6.6	2,000	58.0	51.4	88	72	2.0				
9. L. G., bilateral extrapleural	Preop.	8.4	3,350	119.4	111.0	93	89	2.5				
	Postop., right extrapleural	7.1	3,000	116.6	109.0	93	88	2.0	Right Left	150 112	1,175 1,200	45 55
	Postop., left extrapleural	7.9	3,000	117.2	109.3	93	86	2.4	Right Left	230 290	1,025 1,475	41 59

2. Results would indicate that only minimal decrease in function may be expected following this type of surgery.

3. Extrapleural pneumonolysis may be the procedure of choice in those selected cases where conservation of existing pulmonary function becomes a necessity.

REFERENCES

1. Jacobaeus, H. C., Frenekner, P., and Björkman, S.: Some Attempts at Determining the Volume and Function of Each Lung Separately, *Acta med. Scandinav.* 79: 174, 1932.
2. Gebauer, P. W.: A Catheter for Bronchspirometry, *J. THORACIC SURG.* 8: 674, 1939.
3. Zavod, W. A.: Bronchspirometry. 1. Description of the Catheter and the Technique of Intubation, *J. THORACIC SURG.* 10: 27, 1940.
4. Björkman, S.: Bronchspirometriska studier vid pneumothorax, *Nord. med. tidskr.* 10: 1964, 1935.
5. Vaccarezza, R. F., Soubrie, A., and Rey, J. C.: Functional Study of Each Lung Before and After Extrapleural Pneumothorax, *Rev. panam. med. y Cir. torax.* 1: 141, 1948.

case there was a significant rise in the M.B.C. after Lucite plombage though bronchspirometry showed a decrease in oxygen consumption and ventilation of the collapsed side (Case 5). This is again interpreted⁴ as indicating the inherent reserve of the uncollapsed side which responds with greatly increased function when greater demands are placed on it by collapse of the contralateral lung.

Although the statistical validity of such a small series is naturally subject to question, we have nonetheless determined the percentage decrease which was found following surgery in three of the tests (M.B.C., V.C., and oxygen consumption) used to evaluate pulmonary function.

In the total of nine cases studied, the M.B.C. was decreased by an average of 5.9 per cent (3.02 liters per minute) in seven cases, while two revealed an increase of 2 per cent (1 liter per minute) and 14 per cent (13.7 liters per minute) respectively over the preoperative figures. Bronchspirometric determination of oxygen consumption on the operated side revealed four cases with an average decrease of 8.7 per cent, while four showed an increase of 5.6 per cent. Vital capacity was decreased in eight cases by an average of 12 per cent with one case showing a postoperative increase of 3 per cent.

DISCUSSION

A recent survey of the literature reveals the presence of only one article devoted to a study of the comparative pulmonary function before and after surgical extrapleural pneumonolysis. In 1948, Vaccarezza and associates⁵ reported the results of a functional study of this type carried out on a series of ten patients with pulmonary tuberculosis. His results showed an average decrease in the vital capacity of the collapsed lung of 28 per cent and a 24.3 per cent decrease in the oxygen consumption. As previously noted, the absolute loss of function in our series of cases was of minimal degree. The discrepancy between the figures obtained by Vaccarezza and ourselves can readily be explained if the roentgen reproductions and the clinical data which is supplied in the body of his article are examined. It at once becomes apparent that the extrapleural stripping and resultant pneumothorax space produced by the South American group was greater than in any of the cases in our series, so that one would logically expect the greater collapse to produce a more marked decrease in function. In addition, the extrapleural space in Vaccarezza's series was maintained with air under atmospheric or positive pressures which would in itself further reduce the expansion of the lung in inspiration. Thus, the two series are not comparable in that our specific aim was to produce a surgical collapse of only sufficient degree to control the disease process while at the same time sparing the existing pulmonary function in those selected patients who revealed markedly reduced total function prior to surgery.

SUMMARY

1. Nine patients with pulmonary tuberculosis are presented upon whom pulmonary function was studied before and after surgical extrapleural pneumonolysis with Lucite plombage.

In its superior and lateral portions it faded out but appeared to be more dense medially and inferiorly. The two lateral projections indicated a considerable tumefaction within the chest wall extending posteriorly from just behind the costocartilages. A possible chondroma or primary neoplasm of the chest wall was diagnosed radiologically.

One month later, in May, the patient was admitted to the hospital for the purpose of biopsy to determine the exact nature of the growth. On admission, her complaints were the lump as described, constipation, and undue fatigue for the previous two months. She also gave a history of amenorrhea for the previous two months. She had no particular complaints referable to the respiratory system. General physical examination was negative except for the mass which clinically was situated in the anterior chest wall just below the medial end of the left clavicle and lateral to the sternal border overlying the first and second ribs. It measured approximately 5 by 3 by 1 cm. The mass was firm and solidly fixed to the chest wall but the skin was freely moveable over it. It was slightly sensitive to palpation. Clinical examination of the lungs was entirely negative. In addition, her breasts were slightly enlarged, engorged, and more tender than usual and pelvic examination disclosed an enlarged gravid uterus. The urine examination was negative, the Kahn and Wassermann blood serology tests were negative, and the hemoglobin was 74 per cent. In order to further clarify the intrathoracic portion of the tumor a pneumothorax of 600 c.c. of air was induced and roentgen-ray study of the chest showed that the mass was firmly related to the chest wall anteriorly, but in the lateral view no pneumothorax line was visible, which suggested that the lung might be adherent to the mass in this area. The association of lung tumor and pregnancy concomitantly necessitated that the possibility of a chorioepithelioma with lung metastasis be ruled out. This was excluded to our satisfaction by assay of the urinary gonadotropins which were reported within a normal range. A punch needle biopsy of the tumor on pathologic section failed to show any tumor cells. A direct biopsy was then carried out. At that time the tumor mass was found to be well encapsulated and a small wedge of tissue submitted for pathologic study showed a fibroma which was not histologically malignant, but the pathologist warned that such a lesion is often progressive, locally invasive, and may readily become more active.

The nature of the tumor was explained to the patient and surgical excision was advised. However, she was extremely anxious to carry her pregnancy through to a successful termination, so much so that she insisted and a compromise was reached whereby the progress of the tumor was followed by monthly examinations until her pregnancy was completed, at the end of which time she was to be readmitted for surgical extirpation. On Dec. 22, 1948, she was delivered of a normal, healthy, female child with no pre- or postnatal complications (Figs. 1 and 2).

She was readmitted to the surgical service on Jan. 29, 1949. During the last two months of her pregnancy the mass had increased in size very markedly. On this admission she complained of a dull pain in the left upper chest posteriorly and in the left axilla, a nonproductive ticklish cough for the past three weeks, and some shortness of breath on exertion. The most striking finding on physical examination was the mass itself which had increased greatly in size and on this admission now measured 10 cm. in the vertical diameter and 8 by 8 cm. in width and depth. The urine examination was negative and the hemoglobin was 82 per cent. Roentgen examination of the chest reported a marked progression since the last examination demonstrating a large soft-tissue tumor mass now occupying the space of the left upper lung lobe and obliterating the latter and now also probably involving the left lung root as well as extending through the thoracic wall. The mass completely masked the upper mediastinum extending from the apex to the level of the fourth rib anteriorly (Fig. 3).

On Feb. 4, 1949, under general intratracheal nitrous oxide and ether anesthesia, excision of the tumor was successfully carried out. A transverse incision was made directly over the tumor mass extending from the left lateral border of the sternum overlying the second rib to the midaxillary line. The skin flaps were freed upward and downward for retraction. The pectoralis muscle groups were stretched tightly over the mass and these were divided in the direction of their fibers and retracted. The tumor mass then presented directly. It appeared to be well encapsulated and was of soft consistency, with some areas of firmness.

BENIGN FIBROMA OF CHEST WALL

REPORT OF A CASE

S. G. BAXTER, M.D., AND D. D. MUNRO, M.D.
MONTREAL, QUEBEC

THE occurrence of primary intrathoracic neoplasms seems to be on the increase, more apparent than real, as indeed are most neoplastic growths, and for essentially the same reason. This is mainly due to the employment of modern diagnostic methods, which fact is particularly applicable in the case of chest tumors because of the increasing widespread practice of mass roentgen-ray examination of the chest and the "awareness" of the present-day practitioner of these relatively silent but dangerous growths.

The most important and most frequent intrathoracic tumors are of pulmonary or bronchiogenic origin and the copious literature on this type is testimony of the fact. Second in frequency are mediastinal tumors and least frequent, or comparatively rare, are the neoplasms arising from the wall of the thorax and extending into the thoracic cavity.

Metastatic neoplasms of the bony thorax are, of course, frequent, but primary tumors arising from the connective tissue of the chest wall are rare. Intrathoracic fibroma is reported as an extremely rare tumor and up to October, 1941, Blades¹ found only 32 cases of mediastinal fibroma in the world literature. Harper,² in 1939, was able to find only 9 recorded instances of intrathoracic fibroma arising from the chest wall.

Intrathoracic fibromas may arise from the structures of the mediastinum or from any tissue of the thoracic wall or vertebrae. In many instances it is difficult to determine whether an intrathoracic tumor arises from the mediastinum or the chest wall, particularly when the growth is large and is adherent to both these structures.

We wish to report the successful surgical removal of a rather unusual benign tumor of the chest wall in that it was both intrathoracic and extrathoracic in location, its origin was undoubtedly from the chest wall, and it exhibited a propensity for very rapid growth during the period of gestation.

REPORT OF A CASE

A 27-year-old housewife first presented herself at the out-patient clinic in April, 1948, complaining of a slightly tender swelling beneath the left clavicle which was first noticed about two months previously. Since that time it had increased slightly in size and caused very little discomfort. Physical examination was completely negative except for a slight swelling beneath the medial portion of the left clavicle which was firm, seemingly fixed to the chest wall, and slightly tender to touch. Roentgen examination of the chest showed an unusual soft tissue shadow present along the left parasternal border anteriorly in the situation of the first and second rib costocartilages on the left side. The mass had a clear-cut border and appeared to be within the thoracic inlet. The right lung field was clear. Tomographic studies at the same time reported a mass unquestionably separate from the mediastinum. Its position was immediately posterior to the first and second rib costocartilages.

From the Department of Surgery, Royal Victoria Hospital, and McGill University.
Received for publication Sept. 22, 1949.

The patient's wife, of Irish and German parentage, and eleven children had always been in good health. One child died at 17 years of age of a ruptured appendix. The family had routine x-ray examinations of the chest because of possible tuberculous contact. The numerous cervical ribs were ob-

Fig. 1.



Fig. 2.

Fig. 1.—H. W. (father), aged 46 years.

Fig. 2.—A. W. (mother), aged 42 years.

served and further studies made. There are four grandchildren, but the only one who was examined roentgenographically had no cervical ribs, so it is not possible to trace the familial tendency beyond the first generation. This is summarized in Table I and shown in Figs. 1 through 7.

FAMILIAL INCIDENCE OF CERVICAL RIBS

ROBERT K. PURVES, M.D., AND PAUL H. WEDIN, M.D.
WICHITA, KAN.

ALTHOUGH the occurrence of cervical rib (or ribs) is relatively common, about $\frac{1}{2}$ per cent according to some studies, in that portion of the literature on cervical ribs which we have had an opportunity to examine the familial tendency is rare. Thompson,¹ in 1908, reported a family of five generations in which seven out of sixty-four members were affected with atrophy of the hand muscles. It was his opinion that this was due to cervical ribs. He had only two members of the family examined roentgenographically and they both showed cervical ribs, so it is not entirely conclusive. Weber² presented a case of cervical ribs with atrophy of the hand muscles in which a brother and sister had asymptomatic, bilateral cervical ribs. Other authors^{3, 4} have discussed the embryology and comparative anatomy and have given theories to account for the formation of cervical ribs. These features will not be discussed here.

HISTORY

H. W., a white man of German extraction, aged 46 years, was first seen in the Sedgwick County Hospital and Clinic in April, 1947, complaining of cough, weight loss, hemoptysis, and general malaise. Tuberculosis was suspected and a chest film ordered. This showed a minimal fibrotic process, probably tuberculous, involving both apices. An incidental finding was a cervical rib on the right. His father was alive but refused to be examined.

TABLE I

RELATION	AGE (YR.)	DATE OF ROENTGENO- GRAM	SIDE OF RIB	OTHER ANOMALIES
Father	46	April, 1947	Right	Sacralization of fifth lumbar vertebra
Mother	42	Sept., 1947	Left	Ununited transverse process first lumbar vertebra, left
Son	1	Sept., 1947	Bilateral	Transitional fifth lumbar vertebra
Daughter	4	Sept., 1947	Bilateral	Posterior neural arch defect in transitional fifth lumbar vertebra
Daughter	7	Sept., 1947	Bilateral	Sacralization of fifth lumbar and neural arch defect
Son	8	Sept., 1947	Bilateral	Sacralization of fifth lumbar vertebra
Daughter	12	Sept., 1947	Bilateral	Transitional fifth lumbar vertebra and neural arch defect
Son	16	March, 1948	None	Spinal bifida occulta
Daughter	20	Feb., 1949	None	None
Daughter	21	April, 1949	None	None
Daughter	22	Feb., 1949	None	None
Daughter	25	Feb., 1949	None	None
Daughter	27	None	None	None
Granddaughter	4	Feb., 1949	None	None

Received for publication Oct. 5, 1949.

From the Chest Clinic of the Sedgwick County Hospital and Clinic.

It is interesting to note that the father has a right unilateral rib and the mother a left unilateral rib and all of the children with cervical ribs have them bilaterally. In the young children we looked specifically for the rudimentary ribs because they may have easily escaped detection. If, as is thought, growth

Fig. 5.

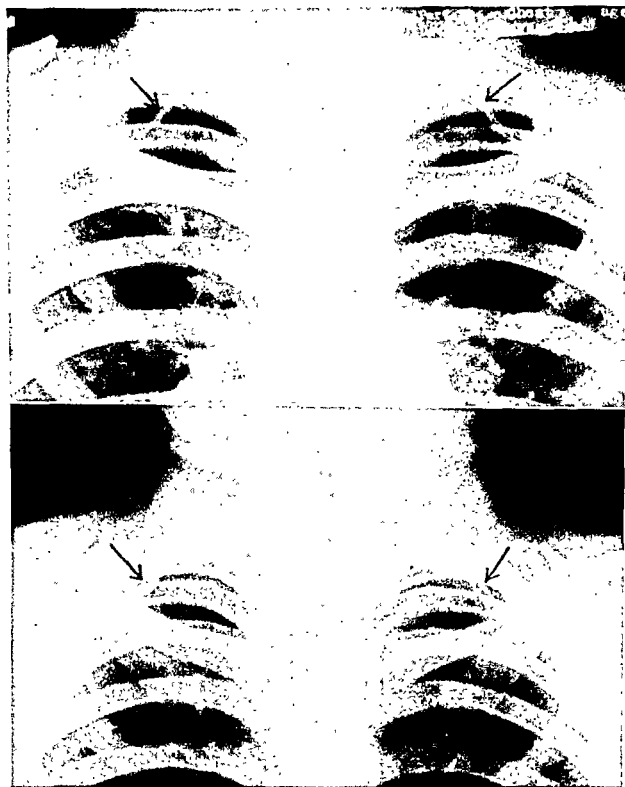


Fig. 6.

Fig. 5.—N. W. (daughter), aged 7 years.

Fig. 6.—E. W. (son), aged 8 years.

occurs from an epiphysis at the apex of the rib, then these rudimentary ribs in the children will probably continue to enlarge and elongate.

Even though the father and all adult members of the family make their living by housework or unskilled labor, none of them have had any symptoms

The possibility of rudimentary first ribs as ruled out by having x-ray films of the entire spine. In this manner twelve ribs could be counted on one side and thirteen on the other in the mother and father. The children who were affected had thirteen bilaterally. As Table I shows, each member of the

Fig. 3.



Fig. 4.

Fig. 3.—W. E. W. (son), aged 1 year.

Fig. 4.—K. W. (daughter), aged 4 years.

family having cervical rib also has some other anomaly of the spine. There are no gross anomalies outside the vertebral column. Adson⁴ and Bassoe⁵ have mentioned other anomalies as a frequent occurrence in cases of cervical rib. There is no scoliosis in any of these cases even though this is reported by Hadley as frequently associated with cervical ribs, particularly if unilateral.

*Normal Development of the Respiratory Tree.*³³—The earliest evidence of the future respiratory tree, the lung bud, is manifest when the foregut measures about 3 mm. The laryngotracheal groove appears in the floor of the foregut just caudal to the pharyngeal pouches. The more caudal end of this anlage gives rise to the lung bud, which begins to bifurcate at about the 4 mm. stage, thus forming the primary bronchi. By further budding the plan of the final pulmonary lobes is already evident at the 7 mm. stage. By the 10 mm. and 14 mm. stages, secondary branchings of the bronchi are defined.

Sequestration of Pulmonary Tissue.—The size of the sequestered mass will depend on the embryologic stage at which sequestration occurred. For instance, sequestration at the 4 mm. stage is likely to produce agenesis of a lung, but sequestration at the 14 mm. stage will probably affect only a lobe or portion of a lobe.

In an attempt to give a reasonable explanation for bronchopulmonary sequestration, Pryce examined the theories regarding the origin of lower accessory lungs. He termed the latter condition "extralobar sequestration," and the former "intralobar sequestration." In both conditions a branch from the aorta supplied the sequestered tissue. In the accessory theory lower accessory lungs are regarded as developments which result from the potentiality of some parts of the foregut to give rise to respiratory tissue. The blood supply from the aorta would be a secondary result of their development. According to the fraction theory, sequestration takes place through such mechanisms as (1) amputation by the developing aortic arch, (2) closure of the pleuroperitoneal iter, (3) deepening of pleural fissures, or (4) by embryonic adhesions. The sequestered tissue, it is said, would then acquire a blood supply in its new situation. This view conforms to the ideas of Beneke³⁴ who stated that a displaced organ easily acquires a blood supply from the region in which it lies in the embryonic body.

However, neither of these theories can be accepted, because in both the blood supply from the aorta is regarded as a factor which is secondary to the existence of the sequestered pulmonary tissue. But, in true fact, an anomalous artery from the aorta sometimes supplies blood to normally connected pulmonary tissue, in which case it cannot possibly be regarded as a secondary event.¹⁵ This is the basis for Pryce's view that the anomalous artery, while developing, may produce sequestration of a portion of pulmonary tissue.

Rosenthal's case suggests that such an anomalous artery may cause sequestration of a portion of the lower lobe, and then itself become separated from the aorta. This case seems to supply the "missing link" between those cases of sequestration obviously associated with an anomalous vessel and those cases of development or congenital cysts of the lung in which an anomalous vessel is not evident (Fig. 9).

PATHOLOGIC ASPECTS OF INTRALOBAR SEQUESTRATION

Sequestered Pulmonary Tissue.—The classification suggested by Pryce as to the gross types of tissue encountered in intralobar sequestration, is as follows: (1) a large cyst in the sequestered area, (2) a polycystic mass, and (3)

the persisting vessels can take practically any course and have any connections that could be traced in the original network. But in actuality the final pattern is usually rather uniform. What actually determines the differentiation of some channels into veins and some into arteries is unknown.

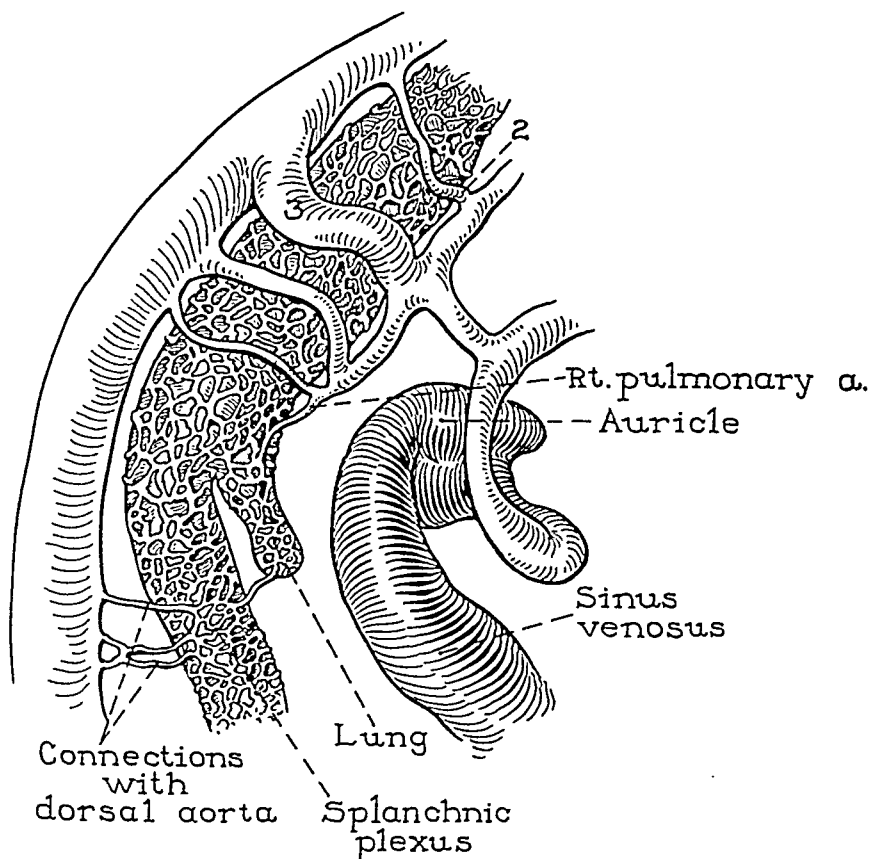


Fig. 1.—Approximately the 4 mm. stage of the foregut which shows the normally developing pulmonary artery and also a few of the connections of the splanchnic plexus with the dorsal aorta; these latter usually atrophy. When they persist and maintain a connection with the pulmonary anlage, an anomalous artery results. Pulmonary venous drainage has been omitted deliberately.

The plexus which concerns this discussion (Fig. 1) is the capillary plexus which embraces the primitive gut, and particularly that part of it in the region of the foregut where the pulmonary anlage develops.²⁷⁻²⁹ This plexus as a whole is the so-called splanchnic plexus of capillaries. It has connections with the dorsal aorta. When the lung bud appears on the ventral surface of the foregut, it carries part of the splanchnic plexus with it. This is the part of the splanchnic plexus which Huntington³⁰ has called the "post-branchial pulmonary plexus." Normally the primitive pulmonary arteries, which develop as angioblastic buds from the region of the aortic sac,^{29, 31, 32} inosculate with the plexus on the lung bud. The connections with the dorsal aorta then atrophy.

The Anomalous Artery.—Fig. 1 indicates that if one connection or more between the dorsal aorta and the postbranchial pulmonary plexus persists, one or more anomalous arteries to the lung will result.

Diameter of Artery.—The diameter of the vessels varied from 2 mm. to 1 cm., and they averaged about 5 mm.

Degenerative Changes in the Artery.—In 5 cases from the literature degenerative changes such as atheroma or medial calcification were mentioned. These changes may be related to the age of the patient and to sepsis.¹ The observation by McDonald and others³⁶ that nonfunction tends toward endarterial changes in vessels supplying a nonfunctioning area would seem to be applicable in cases of bronchopulmonary sequestration when endarteritis is found.

Distribution of Anomalous Artery.—Although an anomalous artery may supply a normal lower lobe, reports indicate that it usually is associated with sequestration of pulmonary tissue.

INCIDENCE OF ANOMALOUS ARTERIES

Anomalous arteries unassociated with any other vascular anomaly have been reported at necropsy in 8 cases and at the time of operation in 26 cases. Knowledge of 9 other cases³⁷ and the addition of 4 new cases in this report makes a total of 47 cases. We are aware of a few additional cases in which other anomalies were associated, but we will not discuss these in this paper.¹⁴⁻¹⁶

TABLE I. AGE OF PATIENTS AT ONSET OF SYMPTOMS AND AT OPERATION AS REPORTED IN THE LITERATURE

AGE (YR.)	NUMBER OF CASES	
	AT ONSET OF SYMPTOMS	AT OPERATION
0 to 9	6	3
10 to 19	7	7
20 to 29	1	5
30 to 39	0	2
40 to 49	0	2
50 to 59	1	1
Total*	15	20

*The discrepancy in totals is due to the fact that in 5 cases the age at onset of symptoms was not mentioned.

At one institution the incidence of anomalous arteries to the lung is reckoned at almost 2 per cent of all pulmonary excisions.³⁸ In the reported cases in which details were given, 15 patients were men and 11 were women, and the sex of 8 is not known. In the surgical cases it often was possible to discover the age at which symptoms first occurred as well as the age at which operation was performed. Table I indicates the distribution in various decades. It is obvious in this small series of cases that the onset of symptoms usually occurred within the first two decades of life. In the 15 cases in which the age of onset was known, the average delay before operation was four years. The shortest period was six months and the longest delay was twenty-one years.

CLINICAL ASPECTS

Symptoms.—The commonest symptoms referred to are productive cough, which becomes aggravated at intermittent periods, fever, pain, and chills,

a sequestered mass which contains bronchi that branch parallel to the artery. Furthermore, in any of these gross types the bronchial or cystic elements may be completely dissociated from the normal bronchial tree or in some communication with the bronchial tree, usually the result of infection or supuration.

Infection of the Sequestered Lung.—Various authors^{1, 5} have drawn attention to the fact that the area of lung supplied by an accessory artery coming off the aorta, is supposedly nonfunctioning. Clagett and McDonald⁵ speculated as to whether or not such a nonfunctioning area would be more likely to become infected than pulmonary tissue which is in physiologic balance. The present views as to the pathology of the sequestered cystic area would not seem to substantiate their speculation as to the cause of infection. In addition, experimental work of Levy and Blalock³⁵ opposed such a mechanism. They transplanted the proximal cut end of the left subclavian artery of mature dogs into the distal cut end of the left pulmonary artery. Although the quantity of oxygen consumed by the lung was found to be small under resting conditions, the lung did remain functional as was proved by increased uptake of oxygen during anoxemia. Respiratory movements on the involved side remained normal. After several months neither the involved pulmonary artery nor the affected lungs showed any noteworthy microscopic alterations. The pressures only a short distance beyond the anastomoses were only one-half those in the systemic arteries.

It would seem that sequestered pulmonary tissue could become infected in various ways: 1. In cases in which there is some communication with the normal bronchial tree, infection could occur via such a communication. 2. In cases of complete sequestration, infection might take place by one of two obvious ways. It might result from direct spread by way of the tissues from an infected, normally connected lung or from hematogenic spread. Pryce and associates² presented an excellent example of the latter in which pulmonary symptoms soon followed osteomyelitis of the femur.

Our report deals primarily with cases of sequestration in which the anomalous artery was the only vascular anomaly in evidence.

Side Involved.—In 18 of the cases reported in the literature the anomalous arteries were on the left and in 10 on the right. One case was that of bilateral arteries; in 5 cases the side was not known.

Type of Artery.—A glance at the titles of articles on the subject will show that most authors write about the anomalous arteries as pulmonary arteries. This cannot possibly have a functional meaning, as pulmonary arteries carry venous blood to the lungs. Calling them "pulmonary" arteries can only indicate that they are arteries going to the lungs. Natucci,¹⁵ in his interesting case of bilateral anomalous arteries in which there was an associated patent ductus arteriosus but in which the lungs were normal, described them as bronchial arteries. Pryce found the anomalous arteries to be histologically more like pulmonary arteries in that they were elastic rather than like systemic arteries which are muscular.

lungs. It is well to bear in mind that the presence of a sequestered, usually cystic, area does not in itself represent a manifestation of the disease. "It is suppuration that makes the disease."⁴⁰ Case 3 is a good example of this, the presence of a cyst having been a fortuitous discovery. As in the surgical cases from the literature, the patients were mainly in the younger age groups and the left side was involved predominantly. A number of wrong diagnoses both clinical and roentgenologic had been made before the correct diagnosis was derived. This was also reflected in the fact that 4 of the 5 patients had been hospitalized at some time before entering the clinic, and 2 of them had undergone unsuccessful operations.

TABLE II. DATA CONCERNING FIVE PATIENTS HAVING ANOMALOUS PULMONARY ARTERIES

	CASES				
	1	2	3*	4	5
SEX	M	F	F	F	F
Age (yr.)					
At onset of symptoms	16	27	18	5	2
At operation	17	30	21	27	34
Side	L	L	L	R	L
Relation of origin of artery to diaphragm	Above	Above	Above	Below	Above
Size of artery	1 cm.	1 cm.	5 mm.	6 mm.	7 mm.
Symptoms	Pain, cough, purulent sputum, fever, chills	Cough, purulent sputum, chills	Fever, chills, syncope, dyspnea on exertion	Cough, purulent sputum, fever	Pain, cough, purulent sputum, fever, chills, dyspnea on exertion
Pathologic group (Pryce)	Dilated bronchi	Polycystic	Large cyst	Large cyst	Polycystic

*This patient had no symptoms until after accidental discovery of cyst during routine physical examination.

Gross Pathologic and Histologic Features.—In our 5 cases the anomalous artery entered the lung in the region of the lower one-half of the pulmonary ligament, usually near the lower border of the lung and fairly far posteriorly. The distribution of the artery could be determined only sketchily in Cases 1, 2, and 3 (Figs. 2, 3, and 4). In Case 4 roentgenograms of the removed lobe after the injection of opaque oil apparently outlined its distribution accurately (Figs. 5 and 6). The anomalous artery seemed to have confined its course to the considerable portion of the lobe which was diseased, that is, sequestered. Unfortunately, the normal pulmonary artery was not injected with oil subsequently so as to determine whether or not its ramifications anastomosed with those of the anomalous artery. Such was the situation in Natucci's case of bilateral anomalous artery, in which pulmonary tissue was normal. In Case 4 subsequent cutting of the tissue into multiple slices made it possible to follow the gross distribution of the artery in the sequestered tissue, to which it seemed to remain confined (Fig. 7). In Case 5 opaque oil was

Diagnosis.—Diagnoses frequently made are pneumonia, empyema, and bronchiectasis. Roentgenograms usually indicate cystic conditions or bronchiectasis. Bronchography was mentioned in 7 cases in the literature. Of these, 2 patients showed bronchiectasis by this method of examination. It is of interest that the other 5 patients did not show evidence of connection between the pathologic area and the bronchial tree.

Bronchoscopy will often reveal a purulent discharge into the bronchi of the lower lobes. If other findings suggestive of sequestration are noted, the latter feature may be reckoned as additional evidence.

Studies of Sputum.—Kirklin³⁹ has pointed out that active tuberculosis is an associated condition in not more than 5 per cent of cases of congenital cysts of the lung. In none of the cases of sequestration of the lower lobe has such a finding been reported.

In view of the fact that a small percentage of bronchiogenic cysts become malignant, the possibility of finding malignant cells in the sputum of patients having sequestration must be borne in mind.

The conditions most commonly entering into the differential diagnosis are acquired bronchiectasis, pneumonia and bronchopneumonia, empyema, and diaphragmatic hernia.

Treatment.—Antibiotic substances such as penicillin and sulfonamide drugs may temporarily improve the acute febrile conditions of the patient. An incorrect diagnosis of empyema may be the cause of repeated aspirations and drainage operations, none of which will be effective in curing the patient. It is evident from the nature of the condition that only surgical extirpation of the diseased lobe can cure intralobar sequestration and its complications. Anomalous arteries are, however, an operative hazard. In 3 cases in the literature,^{4, 17, 22} lobectomies which were apparently going well were fatally complicated by hemorrhage from the anomalous artery. Haight³⁷ warned that in operating on cystic lower lobes the surgeon should always be on the lookout for anomalous arteries.

MATERIAL AND METHODS

The pathologic material used in this study was obtained as a result of lobectomies on 5 patients at the Mayo Clinic during the years 1944 to 1949. All specimens were lower lobes which showed the presence of intralobar sequestration associated with large anomalous arteries. As stated before, 1 case has been reported previously in the literature.⁵ The method of examination of tissue will be described with the observations on it.

OBSERVATIONS AND COMMENTS

The data concerning our cases are presented in tabular form (Table II).

In these 5 cases observations on the main points of interest were in agreement with those in the surgical cases from the literature. The symptoms were typical, that is, productive cough, chills, fever, and pain. All of these symptoms were intermittent as is usually true of congenital cystic disease of the

anastomoses between the branches of the anomalous artery and those of the pulmonary artery, conformed to the findings in Natucci's case.

The question arises whether or not degenerative changes in anomalous arteries are due to pressure or to nonfunction. The anomalous artery in the case reported by Batts⁸ showed medial calcification and endarteritis. The

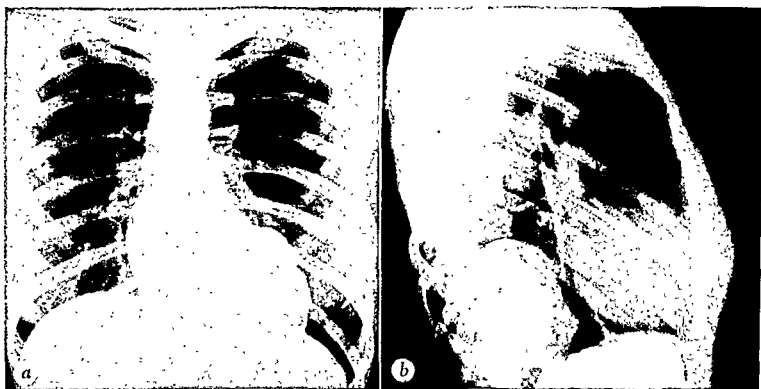


Fig. 4 (Case 3).—*a* and *b*, Unicaneral cyst indicated in left lower lobe. A diagnosis of encapsulated empyema was entertained in this case. At lobectomy the involved lobe was seen to receive a large anomalous artery from the aorta.

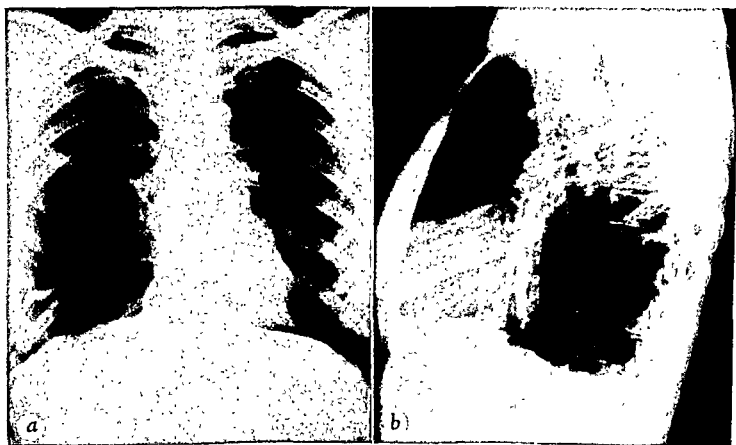


Fig. 5 (Case 4).—*a* and *b*, Large cystic area shown in right lower lobe. Patient arrived at clinic having undergone unsuccessful "drainage" operations on three previous occasions.

not injected into the vessels, but the injection of water into the normal pulmonary artery or the anomalous artery, revealed the presence of substantial communication between the 2 vessels, probably in the precapillary region. Multiple slices proved that the branches of the anomalous artery ran in close relationship to the walls of the cystic spaces (Fig. 8). The finding of obvious

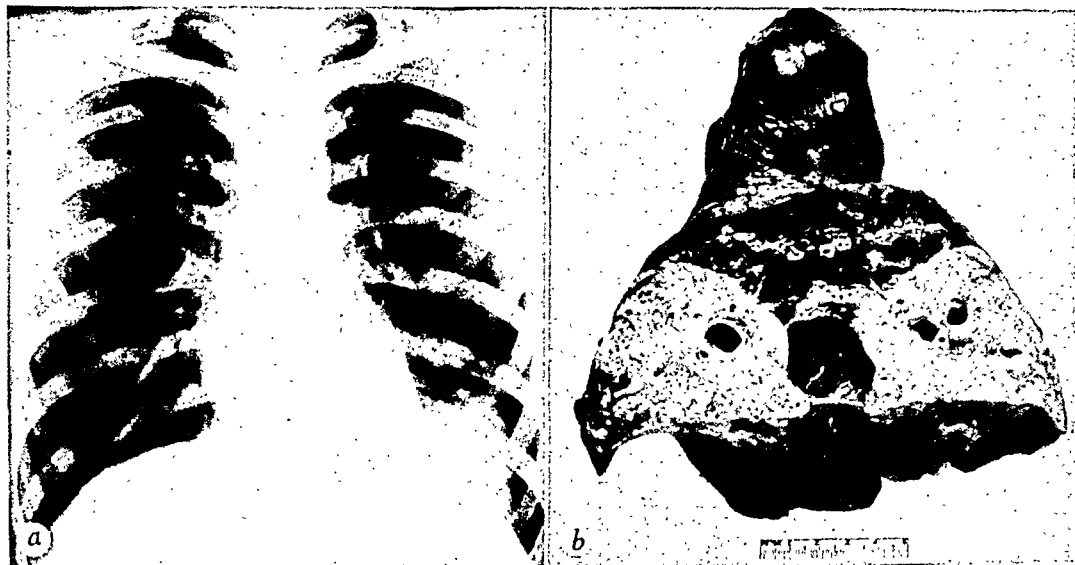


Fig. 2 (Case 1).—*a*, Opacity in the region of the left lower lobe. *b*, Gross specimen of removed left lower lobe. Sequestered part of lobe (white) contains some well-indicated dilated bronchi. These bronchi had no connection with the normal bronchial tree. Anomalous vessel was cut longitudinally and can be seen near the outer edge of each half of the sequestered area.

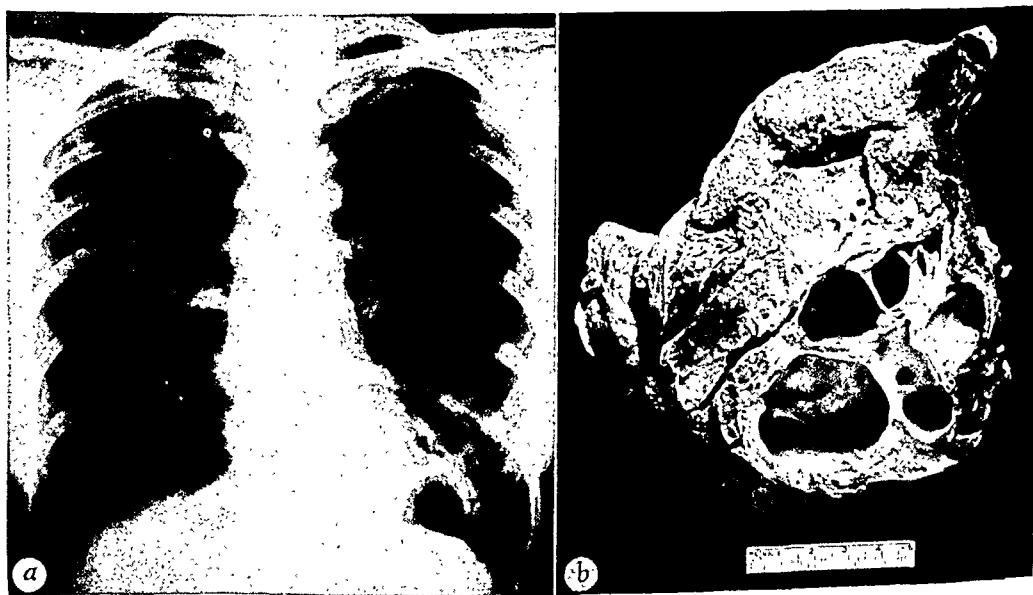


Fig. 3 (Case 2).—*a*, Multiple cystic areas shown with levels of fluid in region of left lower lobe. *b*, Removed left lower lobe in which multiple cysts are demonstrated and branches of anomalous artery can be seen distributed in the walls of the cysts.

artery supplied normal pulmonary tissue. Those areas of lung not supplied by the artery did not reveal the same changes in the vessels. It may be reasoned, as Batts reasoned, that pressure was the cause of the degenerative

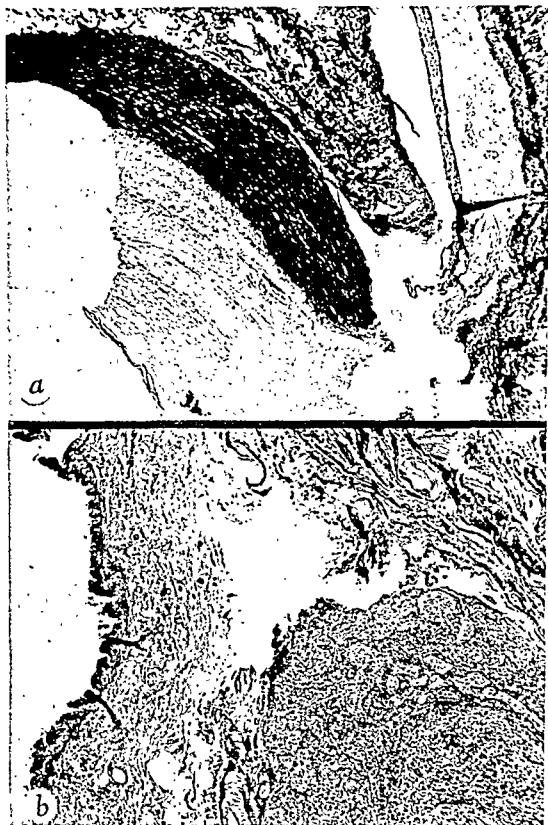


Fig. 7 (Case 4).—*a*, Anomalous vessel stained with van Gieson's stain ($\times 35$). Elastic nature of artery easily seen. Note evidence of endarteritis. *b*, Portion of sequestered lung indicating lining of cyst, and unpigmented lymph node which was situated subpleurally at entrance of anomalous artery into lobe (hematoxylin and eosin, $\times 35$).

changes in the vessel in this case. But it may be said with equal truth that the area supplied by the artery, carrying arterial blood, was nonfunctional under average conditions of oxygen demand, and that this was the cause of the degenerative changes in the vessel. The work of Levy and Blalock discussed earlier in the paper suggested that transplanting the subclavian artery

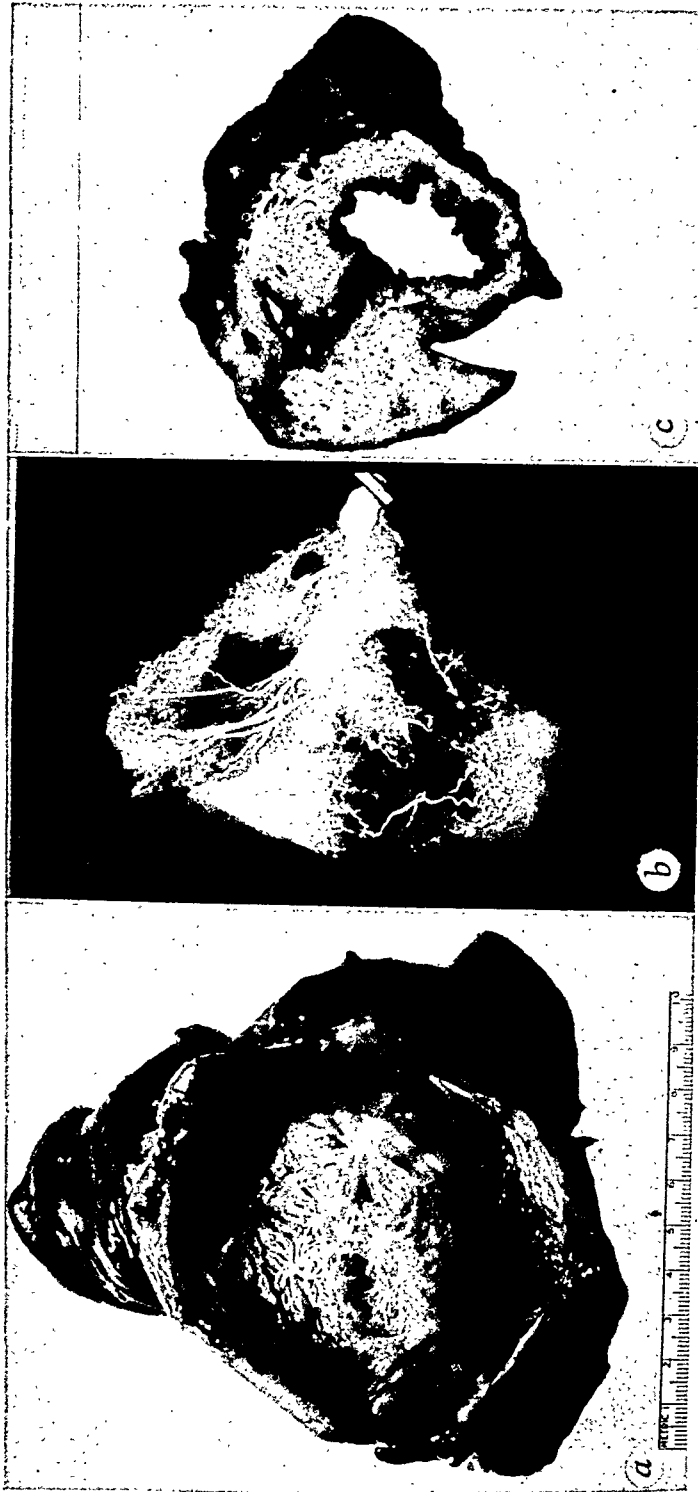


Fig. 6 (Case 4).—*a*, Gross specimen of right lower lobe. *b*, Distribution of radiopaque oil injected into anomalous artery which supplied the sequestered portion of the lung. *c*, Three millimeter slab of the removed lobe which indicates sequestered area. Because of the total lack of pigmentation and aeration the area appeared white. Compare this with normally aerated portions (dark).

cases this can probably be regarded as good evidence that the area of lung drained by these nodes was not at all functional, that is, it was totally sequestered. In Case 2 microscopic study revealed a few small lymph nodes in relation to the larger arterial branchings in the sequestered area. Only one of these contained a small amount of pigment. In the 2 cases in which the intact lower lobes could be studied by the multiple-slice method, it was surprising to note the large proportion of the lobe which consisted of unaerated, unpigmented tissue. This comprised possibly 70 to 80 per cent of the lobe. Shrinkage due to formalin no doubt makes this figure somewhat higher than the true condition would be, owing to aerated normal parts shrinking more than the solid and unaerated parts.

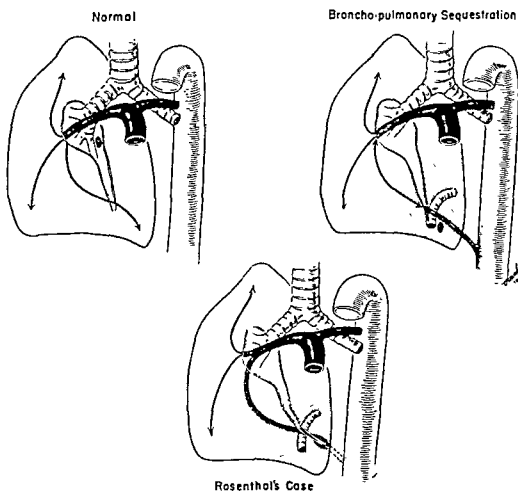


Fig. 9.—Is it possible that Rosenthal's case indicates a link between bronchopulmonary sequestration and some cases of congenital cystic disease of the lungs?

SUMMARY AND CONCLUSIONS

The concept of intralobar bronchopulmonary sequestration and its relationship to associated anomalous arteries from the aorta is a concept of cause and effect. The reasoning of Pryce that the anomalous artery is a primary factor and that in many cases in which it occurs it causes sequestration of a portion of the lower lobe is logical and is substantiated by the pathologic picture. It is a more reasonable way of regarding this condition than the argument which puts sequestration first and the anomalous vessels second.

Intralobar sequestration of itself is not a new entity. Most congenital cysts of the lung, wherever situated in its substance, may be regarded as examples of sequestration. In situations other than the lower lobes, anomalies

into the pulmonary artery did not result in any great rise of pressure in the pulmonary circulation in dogs. It would, therefore, seem reasonable that non-function rather than pressure might have been the cause of endarteritis in Batts' case. Observations by McDonald, Harrington, and Clagett³⁶ tend to support this view. This is, of course, only conjecture.

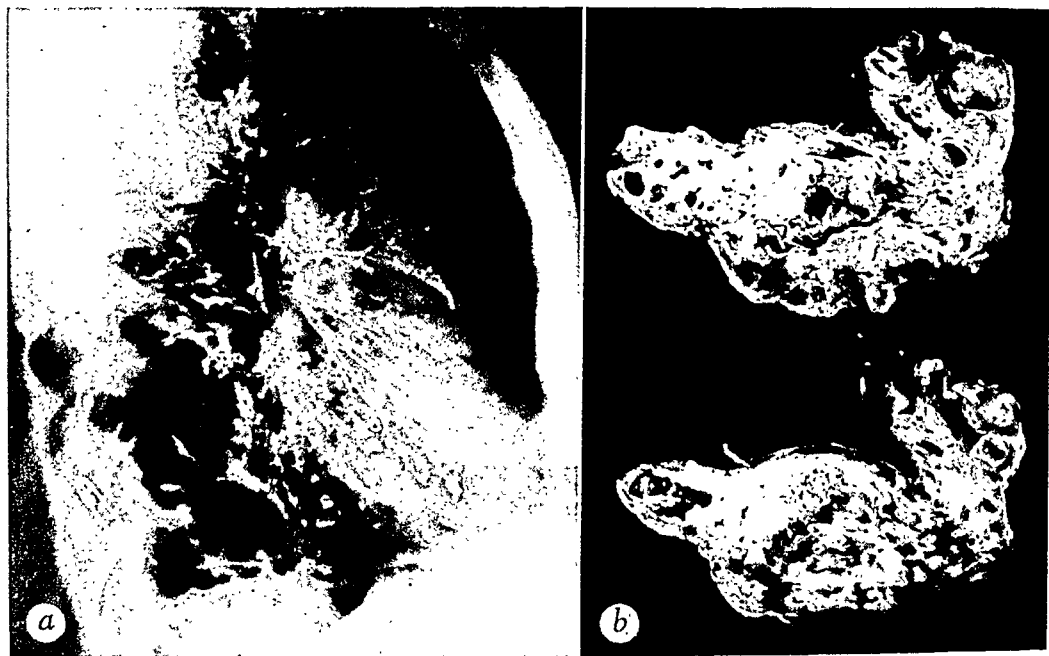


Fig. 8 (Case 5).—*a*, Left lateral bronchogram. Note posterior basal segment did not fill with opaque oil. This is the sequestered cystic area. *b*, Slabs from specimen removed at lobectomy. Note sequestered area (white) which is unpigmented and unaerated. Also, large anomalous artery and associated unpigmented, subpleurally situated lymph node.

Studies of pressure and analyses of gas by means of catheterization of the pulmonary arteries of patients suspected of having anomalous arteries to the lower lobes of lungs would be helpful in reconstructing the physiologic and hemodynamic picture in such lobes. Intravenous studies using radiopaque media with the aid of roentgenoscopy would be of further help. Moreover, after surgical removal of affected lobes, studies by means of injections of radiopaque plastic materials such as Vinylite mixed with iodoform would give accurate radiographic evidence of the structural status of the lobe, and subsequent corrosion of the specimen would leave true casts of the prevailing anatomic conditions. These factors would then be better correlated with pathology, aiding in the interpretation of histologic material. Such studies have been made in other pulmonary conditions by Lindskog⁴¹ and his associates and would supply the necessary background for the suggested studies on sequestration.

In each of 3 cases herein described, a lymph node of fair size was found situated subpleurally in the region where the anomalous artery entered the lobe. In Case 1 evidence of pigmentation in the node was slight. In Cases 4 and 5 the nodes did not show any carbon pigmentation. In the latter two

INDEX TO VOLUME 19

A

- Abbott, Osler A. Discussion of acquired nonmalignant esophagotracheo-bronchial fistula, 556
- Experiences with surgical resection of human carina, tracheal wall, and contralateral bronchial wall in cases of right total pneumonectomy, 906
- Abdominal and right thoracic approach to lesions of middle and upper thirds of esophagus, combined (Kent and Harbison), 559
- Acidosis during thoracic surgery (Beecher and Murphy), 50
- Actinomyces in putrid empyema (Rabin and Janowitz), 355
- Adams, Herbert D. Discussion of acquired nonmalignant esophagotracheo-bronchial fistula, 557
- Transthoracic thyroidectomy, 741, 753
- Adams, William E. Discussion of surgical treatment of bronchiectasis, 265
- Discussion of symposium on resection of the esophagus, 539
- (with Bogardus, George M., and Phillips, Francis J.). Bronchiogenic carcinoma. II. Correlation of pathologic characteristics and clinical manifestations in unresectable and unexplored post-mortem lung tumors, 699
- (with Phillips, Francis J., and Basinger, Clair E.). Bronchiogenic carcinoma. I. Pathologic clinical correlative study of full-size mounts from operated carcinomas, 680
- Adelman, Milton H. (with Berman, Robert A., and Touroff, Arthur S. W.). New method of automatic controlled respiration, 817
- Adenocarcinoma of lung, mucocellular papillary; lobectomy, five year follow-up (Osserman and Neuhof), 875
- Adenomatosis, pulmonary alveolar. Cancerous pulmonary adenomatosis, alveolar cell carcinoma of lung, Jagzietke? (Stephens and Shipman), 559
- Adie, George C. (with Childress, William G.). Plasma cell tumors of mediastinum and lung, 794
- Alexander, John. Discussion of chronic "spontaneous" pneumothorax, 195
- Discussion of pulmonary resection for metastatic malignant lesions, 676
- Allbritten, Frank F., Jr. (with Lipshutz, Herbert, Miller, Bernard J., and Gibbon, John H., Jr.). Blood volume changes in tuberculous patients treated by thoracoplasty, 71, 79
- Allen, Lane. Transpleural muscles, 290
- Alveolar cell carcinoma of lung, Jagzietke? cancerous pulmonary adenomatosis; pulmonary alveolar adenomatosis (Stephens and Shipman), 559
- ventilation perfusion relationships after pneumonectomy, pulmonary circulation and (Cournand, Riley, Himmelstein, and Austrian), 80
- American Association for Thoracic Surgery: board of thoracic surgery, 166, 332
- fellowship for research in tuberculosis (James Alexander Miller), 322
- list of officers, 973
- notice of 30th annual meeting, 332
- 31st annual meeting, 973
- program, 30th annual meeting, 652
- tumor registry committee, 332
- Anastomoses, experimental study of blood supply of esophagus and its relation to esophageal (Shek, Prietto, Tuttle, and O'Brien), 523
- Anastomosis, inlying rubber tube after esophageal (Deaton, Postlethwait, Bradshaw, and Williams), 649
- treatment of short esophageal strictures by resection and end-to-end (Tuttle and Day), 534
- Anesthesia in experimental thoracic surgery, apparatus for (Hanlon, Johns, and Thomas), 887
- Anesthetic blocking of vagus, sympathetic, and phrenic at neck in thoracic surgery—its protective action on blood pressure (Soave), 157
- Aneurysm of first part of left subclavian artery (Temple), 412
- Aortic valve, artificial (Campbell), 312
- Apicolysis in thoracoplasty, an evaluation of (Hagn-Meinke), 853
- Apparatus for anesthesia in experimental thoracic surgery (Hanlon, Johns and Thomas), 887
- Arey, J. B. (with DeBaKey, Michael, and Brunazzi, R.). Successful removal of lower accessory lung, 304
- Arteries to lung associated with congenital pulmonary abnormality, anomalous (Bruwer, Clagett, and McDonald), 957
- Arteriovenous fistula of lung (Carswell), 789
- Artery, aneurysm of first part of left subclavian (Temple), 412

Abstracts for presentation of papers for the 1951 meeting must be received for consideration by the Program Committee on or before January 1, 1951. These abstracts should be submitted in *quadruplicate* and addressed to the Secretary of the Association, Dr. Brian Blades, George Washington University Hospital, 901 23rd Street, N.W., Washington 7, D. C. They should not be sent to the Editor. Abstracts received after January 1, 1951, will not be considered.

- Bruwer, Andre (with Clagett, O. Theron, and McDonald, John R.). Anomalous arteries to lung associated with congenital pulmonary abnormality, 957
- Buckles, Maurice G. (with Lawless, Edward C.). Giant-cell tumor of ribs, 438
- Bunch, George H., Jr. (with Coleman, Frank Philip). Acquired nonmalignant esophagotracheobronchial fistula, 542, 558
- Burnett, W. Emory. Discussion of combined abdominal and right thoracic approach to lesions of middle and upper thirds of esophagus, 571
Discussion of resection of esophagus, 540
(with Long, Joan H., Norris, Charles M., and Wester, Mary R.). Bronchospirometric studies in bronchiectasis, before and after lobectomy, 477
- Burroughs, Mider G. (with Valle, Anibal Roberto). Mechanism of healing of lung tissue and its reaction to different suture materials, 324
- C
- Campbell, Darrell A. Reconstruction of anterior thoracic wall, 456
- Campbell, J. Moore. Artificial aortic valve, 312
- Carcinoma, bronchiogenic. I. Pathologic clinical correlative study of full-size mounts from operated carcinomas (Phillips, Basinger, and Adams), 680
II. Correlation of pathologic characteristics and clinical manifestation in unresectable and unexplored post-mortem lung tumors (Bogardus, Adams, and Phillips), 699
- Cárdenas, Consuelo (with Staines, Enrique). Streptomycin plus alkalinization of pleural cavity in treatment of tuberculous empyema, 891
- Carswell, James, Jr. Arteriovenous fistula of lung, 789
(with Kraefft, Nelson H.). Fibrosarcoma of bronchus; report of case diagnosed by bronchoscopy and treated by pneumonectomy, 117
- Cassel, M. A. (with Cunningham, R. T., and Weisel, Wilson). Foregut cyst of mediastinum, 138
- Cervical ribs, familial incidence of (Purves and Wedin), 952
- Chamberlain, J. Maxwell. Discussion of combined abdominal and right thoracic approach to lesions of middle and upper thirds of esophagus, 569
(with Ryan, Thomas C.). Segmental resection in pulmonary diseases, 199, 224
- Childress, William G. (with Adie, George C.). Plasma cell tumors of mediastinum and lung, 794
- Chondrosarcoma of posterior mediastinum with hourglass involvement of spinal canal; resection and recovery (Weisel and Ross), 643
- Churchill, Edward D. Discussion of resection of esophagus, 540
- Chylothorax (Meade, Head, and Moen), 709
- Clagett, O. Theron. Discussion of pulmonary alveolar adenomatosis; cancerous pulmonary adenomatosis, alveolar cell carcinoma of lung, Jagzicktef, 602
(with Bruwer, Andre, and McDonald, John R.). Anomalous arteries to lung associated with congenital pulmonary abnormality, 957
(with Grindlay, John H., and Rydell, John R.). Permanent filling of dead space in pleural cavity after pneumonectomy, 391
(with Schmidt, Herbert W., and McDonald, John R.). Broncholithiasis, 226
(with Seiler, Hawley H., and McDonald, John R.). Pulmonary resection for metastatic malignant lesions, 655
- Coello, Alfred J. Transscapular thoracoplasty, 900
- Coleman, Frank Philip. (with Bunch, George H., Jr.). Acquired nonmalignant esophagotracheobronchial fistula, 542
- Congenital bronchoesophageal fistula persistent to adult life, an apparently (Morton, Osborne, and Klassen), 811
pulmonary abnormality, anomalous arteries to lung associated with (Bruwer, Clagett, and McDonald), 957
- Conklin, William S. (with Watkins, Elton, Jr.). Use of Potts-Smith-Gibson clamp for division of patent ductus arteriosus, 361
- Connolly, Maurice R. (with Forsee, James H.). Thoracoplasty and streptomycin in treatment of tuberculous empyema, 877
- Courmand, Andre. Discussion of broncholithiasis, 245
(with Riley, R. L., Himmelstein, A., and Austrian, R.). Pulmonary circulation and alveolar ventilation perfusion relationships after pneumonectomy, 80
- Cunningham, R. T. (with Cassel, M. A., and Weisel, Wilson). Foregut cyst of mediastinum, 138
- Curry, John J. (with Fuchs, Job E.). Expectoration of a fibrosarcoma, 135
- Curti, P. C. (with Poulsen, Thue). Treatment of simple spontaneous pneumothorax, 145
- Cyst of chest wall lined with bronchial epithelium, accidental (Stenimermann and Duffy), 349
of mediastinum, a foregut (Cassel, Cunningham, and Weisel), 138

- Aufses, Arthur. Discussion of pulmonary resection for metastatic malignant lesions, 678
- Austrian, R. (with Cournand, A., Riley, R. L., and Himmelstein, A.). Pulmonary circulation and alveolar ventilation perfusion relationships after pneumonectomy, 80
- Avery, E. E. Discussion of broncholithiasis, 244
Discussion of transthoracic thyroidectomy, 752
- B
- Bailey, Charles P. Discussion of combined abdominal and right thoracic approach to lesions of middle and upper thirds of esophagus, 570
(with Glover, Robert P., and O'Neill, Thomas J. E.). Surgery of mitral stenosis, 16, 48
- Basinger, Clair E. (with Phillips, Francis J., and Adams, William E.). Bronchiogenic carcinoma. I. Pathologic clinical correlative study of full-size mounts from operated carcinomas, 680
- Baxter, S. G. (with Munro, D. D.). Benign fibroma of chest wall, 944
- Beardsley, J. Murray. Use of tantalum plate when resecting large areas of chest wall, 444
- Beecher, Henry K. (with Murphy, Anna J.). Acidosis during thoracic surgery, 50, 69
- Berman, Robert A. (with Adelman, Milton H., and Touroff, Arthur S. W.). New method of automatic controlled respiration, 817
- Berry, Frank B. Discussion of mesothelial mediastinal cysts, 768
- Black, Harrison. Fibrosarcoma of bronchus, 123
- Blades, Brian B. Discussion of chronic "spontaneous" pneumothorax, 197
- Blood and nerve supply to esophagus (Swenson, Merrill, Peirce, and Rheinlander), 462
pressure, protective action on, anesthetic blocking of vagus, sympathetic, and phrenic at neck in thoracic surgery (Soave), 157
supply of esophagus and its relation to esophageal resection and anastomoses, an experimental study of (Shek, Prietto, Tuttle, and O'Brien), 523
volume changes in tuberculous patients treated by thoracoplasty (Allbritten, Lipshutz, Miller, and Gibson). 71
- Bogardus, George M. (with Adams, William E., and Phillips, Francis J.). Bronchiogenic carcinoma. II. Correlation of pathologic characteristics and clinical manifestation in unresectable and unexplored post-mortem lung tumors, 699
- Boley, James O. (with Kittle, C. Frederick, and Schafer, Paul W.). Resection of intrathoracic "hibernoma," 830
- Bradshaw, H. H. (with Deaton, W. Ralph, Jr., Postlethwait, R. W., and Williams, Robert W.). Inlying rubber tube after esophageal anastomosis, 649
- Brantigan, O. C. Discussion of mitral stenosis, 47
(with Rigdon, H. L.). Sodium tetradecyl sulfate, azochloramide, and sulfanilamide mixture in prevention of empyema following pneumonectomy with contamination, 319
- Brewer, Lyman A., III (with Dolley, Frank S., and Evans, Byron H.). Surgical management of chronic "spontaneous" pneumothorax, 167, 197
- Brodkin, Henry A. Discussion of funnel chest and deformities of thoracic cage, 520
- Bronchial epithelium, accidental cyst of chest wall lined with (Stemmermann and Duffy), 349
- Bronchiectasis associated with anomaly of right pulmonary vein and right diaphragm (Drake and Lynch), 433
before and after lobectomy, bronchspirometric studies in (Long, Norris, Burnett, and Wester), 477
results of surgery in (Lambert), 246
surgical treatment of bilateral (Kérgin), 257
- Bronchiogenic carcinoma. I. Pathologic clinical correlative study of full-size mounts from operated carcinomas (Phillips, Basinger, and Adams), 680
II. Correlation of pathologic characteristics and clinical manifestation in unresectable and unexplored post-mortem lung tumors, (Bogardus, Adams, and Phillips), 699
- Bronchspirometric studies in bronchiectasis, before and after lobectomy (Long, Norris, Burnett, and Wester), 477
- Bronchspirometry in the lateral decubitus position (Rothstein, Landis, and Narodick), 821
- Bronchostenosis, plastic reconstruction of tuberculous, with dermal grafts (Gebauer), 604
- Bronchus, fibrosarcoma of (Black), 123
report of case diagnosed by bronchoscopy and treated by pneumonectomy (Carswell and Kraefft), 117
- Bronfin, Gerald J. (with Dressler, Sidney H., and Grow, John B.). Pulmonary function studies before and after extrapleural pneumonolysis with plombage, 938
- Brown, A. L. Discussion of funnel chest and deformities of thoracic cage, 519
- Brunazzi, R. (with DeBakey, Michael, and Arey, J. B.). Successful removal of lower accessory lung, 304

Evans, Byron H. (with Brewer, Lyman A., III, and Dolley, Frank S.). Surgical management of chronic "spontaneous" pneumothorax, 167

F

Fibroma of chest wall, benign (Baxter and Munro), 944

Fibrosarcoma, expectoration of (Curry and Fuchs), 135

of bronchus (Black), 123

report of case diagnosed by bronchoscopy and treated by pneumonectomy (Carswell and Kræfft), 117

Fistula of lung, arteriovenous (Carswell), 789

persistent to adult life, apparently congenital bronchoesophageal (Morton, Osborne and Klassen), 811

Forsee, James H. Discussion of surgical treatment of bronchiectasis, 268

surgical treatment of round tuberculous pulmonary lesions (tuberculomas), 724

(with Connolly, Maurice R.). Thoracoplasty and streptomycin in treatment of tuberculous empyema, 877

Fox, Robert T. Discussion of pulmonary alveolar adenomatosis; cancerous pulmonary adenomatosis, alveolar cell carcinoma of lung, Jagzietke?, 603

Franklin, R. H. (with Taylor, Sewlyn). Nonspecific granulomatous (regional) esophagitis, 292

Fuchs, Job E. (with Curry, John J.). Expectoration of a fibrosarcoma, 135

Funnel chest and allied deformities of thoracic cage (Lester), 507

G

Gaensler, Edward A. (with Sarnoff, Stanley J., and Maloney, James V., Jr.). Electrophrenic respiration. IV. Effectiveness of contralateral ventilation during activity of one phrenic valve, 929

Garlock, John H. Discussion of combined abdominal and right thoracic approach to lesions of middle and upper thirds of esophagus, 568

(with Som, Max L.). Further observations on packing of mediastinum for esophageal varices, 572, 587

Gault, Edwin S. (with Sano, Machteld E., and Weiss, Edward). Pleural mesothelioma, 783

Gebauer, Paul W. Plastic reconstruction of tuberculous bronchostenosis with dermal grafts, 604

Giant-cell tumor of ribs (Buckles and Lawless), 438

Gibbon, John H., Jr. Discussion of acidosis during thoracic surgery, 69

Gibbon—Cont'd

Discussion of combined abdominal and right thoracic approach to lesions of middle and upper thirds of esophagus, 569

Discussion of resection of esophagus, 540

(with Allbritten, Frank F., Jr., Lipshutz, Herbert, and Miller, Bernard J.). Blood volume changes in tuberculous patients treated by thoracoplasty, 71

(with Healey, John E., Jr.). Intrapericardial anatomy in relation to pneumonectomy for pulmonary carcinoma, 864

Glover, Robert P. Discussion of segmental resection, 222

(with Bailey, Charles P., and O'Neill, Thomas J. E.). Surgery of mitral stenosis, 16

Goldman, Alfred. Discussion of chronic "spontaneous" pneumothorax, 197

Discussion of mediastinal "tuberculoma," 348

(with Segal, J., and Sherman, Leon H.). Thoracoplasty, 270

Gordon, Joseph. Discussion of surgical treatment of bronchiectasis, 265

Grace, A. J. Discussion of broncholithiasis, 244

Discussion of mediastinal "tuberculoma," 347

Discussion of mitral stenosis, 46

Discussion of transthoracic thyroidectomy, 752

Graham, Everts A. Discussion of pulmonary alveolar adenomatosis; cancerous pulmonary adenomatosis, alveolar cell carcinoma of lung, Jagzietke?, 599

Granulomatous (regional) esophagitis, non-specific (Franklin and Taylor), 292

Grindlay, John H. (with Clagett, O. Theron, and Rydell, John R.). Permanent filling of dead space in pleural cavity after pneumonectomy, 391

Grow, John B. (with Dressler, Sidney H., and Bronfin, Gerald J.). Pulmonary function studies before and after extrapleural pneumonolysis with plombage, 938

Guptill, Plimpton. Discussion of mesothelial mediastinal cysts, 768

H

Hagn-Meineke, F. Evaluation of apicolysis in thoracoplasty, 853

Late results in four hundred twenty tuberculous patients subjected to thoracoplasty, 837

Haight, Cameron. Discussion of acquired nonmalignant esophagotracheobronchial fistula, 558

D

- Dailey, J. E. Discussion of funnel chest and deformities of thoracic cage, 519
- Daniel, Rollin A., Jr. Discussion of pneumonectomy followed by immediate thoracoplasty, 505
(with Williams, R. B., Jr.). Leiomyoma of esophagus, 800
(with Williams, R. B., Jr.). Leiomyoma of lung, 806
- Davis, Edgar W. Discussion of broncholithiasis, 244
Discussion of pulmonary alveolar adenomatosis; cancerous pulmonary adenomatosis, alveolar cell carcinoma of lung, Jagziette?, 602
- Day, J. C. (with Tuttle, W. M.). Treatment of short esophageal strictures by resection and end-to-end anastomosis, 534
- Deaton, W. Ralph, Jr. (with Postlethwait, R. W., Bradshaw, H. H., and Williams, Robert W.). Inlying rubber tube after esophageal anastomosis, 649
- DeBakey, Michael (with Arey, J. B., and Brunazzi, R.). Successful removal of lower accessory lung, 304
- Decker, H. Ryerson. Discussion of pulmonary alveolar adenomatosis; cancerous pulmonary adenomatosis, alveolar cell carcinoma of the lung, Jagziette?, 600
Evaluation of extrapleural pneumonolysis and paraffin plombage for chronic pulmonary disease, 629
- Dermal grafts, plastic reconstruction of tuberculous bronchostenosis with (Gebauer), 604
- Diaphragm, temporary high position of (Meyler and Huizinga), 283
- Dolley, Frank S. (with Brewer, Lyman A., III, and Evans, Byron H.). Surgical management of chronic "spontaneous" pneumothorax, 167
- Domm, Sheldon E. Discussion of chronic "spontaneous" pneumothorax, 197
- Drake, Emerson H. (with Lynch, Joseph P.). Bronchiectasis associated with anomaly of right pulmonary vein and right diaphragm, 433
- Drash, E. C. (with Hyer, Harry J.). Mesothelial mediastinal cysts, 755, 768
- Dressler, Sidney H. (with Bronfin, Gerald J., and Grow, John B.). Pulmonary function studies before and after extrapleural pneumonolysis with plombage, 938
- Duffy, Robert W. (with Stemmermann, Grant N.). Accidental cyst of chest wall lined with bronchial epithelium, 349
- Dugan, David J. (with Samson, Paul C., and Heaton, Leonard D.). Mediastinal "tuberculoma," 333

E

- Ellis, Laurence B. (with Harken, Dwight E., and Norman, Leona R.). Surgical treatment of mitral stenosis. II. Progress in developing controlled valvuloplastic technique, 1
- Emerson, E. C. (with Merendino, K. Alvin). Aortoesophagogastric fistula, unusual complication of esophagogastrostomy performed under aortic arch following esophageal resection for carcinoma, 405
- Empyema, actinomycetes in putrid (Rabin and Janowitz), 355
following pneumonectomy with contamination, sodium tetradeceyl sulfate, azochloramide, and sulfanilamide mixture in prevention of (Rigdon and Brantigan), 319
streptomycin plus alkalinization of pleural cavity in treatment of tuberculous (Staines and Cárdenas), 891
thoracoplasty and streptomycin in treatment of tuberculous (Connolly and Forsee), 877
- Esophageal anastomosis, inlying rubber tube after (Deaton, Postlethwait, Bradshaw, and Williams), 649
motility in surgical treatment of megacophagus, role of (Puppel), 371
resection for carcinoma, aortoesophagogastric fistula, unusual complication of esophagogastrostomy performed under aortic arch following (Merendino and Emerson), 405
strictures by resection and end-to-end anastomosis, treatment of short (Tuttle and Day), 534
varices, further observations on packing of mediastinum for (Garlock and Som), 572
- Esophagitis, nonspecific granulomatous (regional), (Franklin and Taylor), 292
- Esophagogastrostomy performed under aortic arch following esophageal resection for carcinoma, aortoesophagogastric fistula, unusual complication of (Emerson and Merendino), 405
- Esophagotracheobronchial fistula, acquired nonmalignant (Coleman and Bunch), 542
- Esophagus and its relation to esophageal resection and anastomoses, experimental study of blood supply of (Shek, Prietto, Tuttle, and O'Brien), 523
blood and nerve supply to (Swenson, Merrill, Peirce, and Rheinlander), 462
combined abdominal and right thoracic approach to lesions of middle and upper thirds of (Kent and Harbison), 559
leiomyoma of (Daniel and Williams), 800

- Klepser, R. G. Discussion of management of chylothorax, 722
- Kraeft, Nelson H. (with Carswell, James, Jr.). Fibrosarcoma of bronchus; report of case diagnosed by bronchoscopy and treated by pneumonectomy, 117
- Kraus, Alvin R. (with Weinberg, Joseph A.). Intercostal incision in transpleural operations, 769

L

- Lam, Conrad R. Discussion of mesothelial mediastinal cysts, 767
- Lambert, Adrian. Discussion of blood volume changes in tuberculous patients treated by thoracoplasty, 79
- Discussion of pulmonary resection for metastatic malignant lesions, 679
- Results of surgery in bronchiectasis, 246, 269
- Landis, Francis B. (with Rothstein, Emil, and Narodick, Benjamin G.). Bronchspirometry in lateral decubitus position, 821
- Lawless, Edward C. (with Buckles, Maurice G.). Giant-cell tumor of ribs, 438
- Lees, William. Discussion of surgical treatment of bronchiectasis, 267
- Leiomyoma of esophagus (Daniel and Williams), 800
- of lung (Williams and Daniel), 806
- Lesions of middle and upper thirds of esophagus, combined abdominal and right thoracic approach to (Kent and Harbison), 559
- pulmonary resection for metastatic malignant (Seiler, Clagett, and McDonald), 655
- Lester, Charles W. Funnel chest and allied deformities of thoracic cage, 507, 522
- Lindskog, Gustaf E. Discussion of mesothelial mediastinal cysts, 768
- Lipshutz, Herbert (with Allbritten, Frank F., Jr., Miller, Bernard J., and Gibson, John H., Jr.). Blood volume changes in tuberculous patients treated by thoracoplasty, 71
- Lobectomy and pneumonectomy in treatment of pulmonary tuberculosis (Sweet), 298
- bronchspirometric studies in bronchiectasis, before and after (Long, Norris, Burnett, and Wester), 477
- five year follow-up; mucocellular papillary adenocarcinoma of lung (Osserman and Neuhoef), 875
- Long, Joan H. (with Norris, Charles M., Burnett, W. Emory, and Wester, Mary R.). Bronchspirometric studies in bronchiectasis, before and after lobectomy, 477
- Lung, mucocellular papillary adenocarcinoma of; lobectomy, five year follow-up (Osserman and Neuhoef), 875

Lung—Cont'd

- successful removal of lower accessory (DeBakey, Arey, and Brunazzi), 304
- tissue and its reaction to different suture materials, mechanism of healing of (Valle and Mider), 324
- Lynch, Joseph P. (with Drake, Emerson H.). Bronchiectasis associated with anomaly of right pulmonary vein and right diaphragm, 433

M

- Mahon, Hugh W. (with Forsee, James H.). Surgical treatment of round tuberculous pulmonary lesions (tuberculomas), 724
- Maier, Herbert. Discussion of symposium on resection of esophagus, 538
- Maloney, James V., Jr. (with Sarnoff, Stanley J., and Gaensler, Edward A.). Electrophrenic respiration. IV. Effectiveness of contralateral ventilation during activity of one phrenic nerve, 929
- Mason, James M., III. Discussion of symposium on resection of esophagus, 539
- Maurer, Elmer R. Discussion of surgical treatment of round tuberculous pulmonary lesions (tuberculomas), 739
- McCorrison, James R. An instrument for passing ligatures, 489
- McDonald, John R. (with Bruwer, Andre, and Clagett, O. Theron). Anomalous arteries to lung associated with congenital pulmonary abnormality, 957
- (with Seiler, Hawley H., and Clagett, O. Theron). Pulmonary resection for metastatic malignant lesions, 655
- McIntyre, J. P. Observations on thoracoplasty during pregnancy, 882
- Meade, Richard H., Jr. Discussion of chronic "spontaneous" pneumothorax, 194
- Discussion of transthoracic thyroidectomy, 750
- (with Head, Jerome R., and Moen, Chester W.). Management of chylothorax, 709, 723
- Mediastinal cysts, mesothelial (Drash and Hyer), 755
- "tuberculoma" (Samson, Heaton, and Dugan), 333
- Mediastinum, and lung, plasma cell tumors of (Childress and Adie), 794
- foregut cyst of (Cassel, Cunningham, and Weisel), 138
- for esophageal varices, further observations on packing of (Garlock and Som), 572
- with hourglass involvement of spinal canal, chondrosarcoma of posterior; resection and recovery (Weisel and Ross), 643

- Hanlon, C. Rollins (with Johns, Thomas N. P., and Thomas, Vivien). Apparatus for anesthesia in experimental thoracic surgery, 887
- Harbison, Samuel P. (with Kent, Edward M.). Combined abdominal and right thoracic approach to lesions of middle and upper thirds of esophagus, 559, 571
- Harken, Dwight E. Discussion of mitral stenosis, 47
(with Ellis, Laurence B., and Norman, Leona R.). Surgical treatment of mitral stenosis. II. Progress in developing controlled valvuloplastic technique, 1
- Harper, F. R. Discussion of management of chylothorax, 720
- Hart, Deryl. Discussion of surgical treatment of bronchiectasis, 268
Discussion of transthoracic thyroidectomy, 751
- Harter, John S. Discussion of acquired non-malignant esophagotracheobronchial fistula, 557
- Head, Jerome. Discussion of bronchiogenic carcinoma, 698
Discussion of broncholithiasis, 243
Discussion of funnel chest and deformities of thoracic cage, 521
(with Meade, Richard H., Jr., and Moen, Chester W.). Management of chylothorax, 709
- Healey, John E., Jr. (with Gibbon, John H., Jr.). Intrapericardial anatomy in relation to pneumonectomy for pulmonary carcinoma, 864
- Heaton, Leonard D. (with Samson, Paul C., and Dugan, David J.). Mediastinal "tuberculoma," 333
- Hernia, roentgenologic manifestations of parasternal omental (Stewart), 399
- Hibernoma, resection of an intrathoracic (Kittle, Boley, and Schafer), 830
- Himmelstein, A. (with Cournand, A., Riley, R. L., and Austrian R.). Pulmonary circulation and alveolar ventilation perfusion relationships after pneumonectomy, 80
- Holinger, Paul. Discussion of funnel chest and deformities of thoracic cage, 521
- Holman, Emile. Discussion of blood volume changes in tuberculous patients treated by thoracoplasty, 79
- Hughes, Felix A., Jr. Discussion of chronic "spontaneous" pneumothorax, 196
Discussion of mesothelial mediastinal cysts, 768
Discussion of segmental resection, 223
- Huizinga, Eelco (with Meyler, L.). Temporary high position of diaphragm, 283
- Hydatid disease, treatment of pulmonary (Susman), 422
- Hyer, Harry J. (with Drash, E. C.). Mesothelial mediastinal cysts, 755

I

- Instrument for passing ligatures (McCorriston), 489
- Intercostal incision in transpleural operation (Weinberg and Kraus), 769
- Iverson, Roland K. (with Skinner, Homer L.). Pneumonectomy followed by immediate thoracoplasty, 491

J

- Jackson, T. L. Discussion of plastic reconstruction of tuberculous bronchostenosis with dermal grafts, 623
- Jagziekte? pulmonary alveolar adenomatosis; cancerous pulmonary adenomatosis, alveolar cell carcinoma of lung (Stephens and Shipman), 589
- Janes, Ernest. Discussion of transthoracic thyroidectomy, 753
- Janes, R. M. Discussion of surgical treatment of bronchiectasis, 266
- Janowitz, Henry D. (with Rabin, Coleman B.). Actinomyces in putrid empyema, 355
- Johns, Thomas N. P. (with Hanlon, C. Rollins, and Thomas, Vivien). Apparatus for anesthesia in experimental thoracic surgery, 887
- Johnson, Julian. Discussion of combined abdominal and right thoracic approach to lesions of middle and upper thirds of esophagus, 570

K

- Kay, Earle B. Discussion of combined abdominal and right thoracic approach to lesions of middle and upper thirds of esophagus, 570
- Kelley, Winfield O. Phrenic nerve paralysis; special consideration of accessory phrenic nerve, 923
- Kent, E. M. Discussion of further observations on packing of mediastinum for esophageal varices, 587
(with Harbison, Samuel P.). Combined abdominal and right thoracic approach to lesions of middle and upper thirds of esophagus, 559, 571
- Kergin, Frederick G. Discussion of transthoracic thyroidectomy, 751
Surgical treatment of bilateral bronchiectasis, 257, 269
- Kisner, Wendell H. (with Reganis, John C.). Pericardial celomic cyst, with symptoms, 779
- Kittle, C. Frederick (with Boley, James O., and Schafer, Paul W.). Resection of intrathoracic "hibernoma," 830
- Klassen, Karl P. (with Morton, Douglas R., and Osborne, Johnston F.). Apparently congenital bronchoesophageal fistula persistent to adult life, 811

- Phillips—Cont'd
 (with Bogardus, George M., and Adams, William E.). Bronchiogenic carcinoma. II. Correlation of pathologic characteristics and clinical manifestation in unresectable and unexplored post-mortem lung tumors, 699
- Phrenic nerve paralysis; special consideration of accessory phrenic nerve (Kelley), 923
- Plastic reconstruction of tuberculous bronchostenosis with dermal grafts (Gebauer), 604
- Pleural cavity after pneumonectomy, permanent filling of dead space in (Grindlay, Clagett, and Rydell), 391
 in treatment of tuberculous empyema, streptomycin plus alkalization of (Staines and Cárdenas), 891
 mesothelioma (Sano, Weiss, and Gault), 783
- Plombage, pulmonary function studies before and after extrapleural pneumonolysis with (Dressler, Bronfin, and Grow), 938
- Pneumonectomy, experiences with surgical resection of human carina, tracheal wall, and contralateral bronchial wall in cases of right total (Abbott), 906
 followed by immediate thoracoplasty (Iverson and Skinner), 491
 for pulmonary carcinoma, intrapericardial anatomy in relation to (Healey and Gibbon), 864
 in treatment of pulmonary tuberculosis, lobectomy and (Sweet), 298
 permanent filling of dead space in pleural cavity after (Grindlay, Clagett, and Rydell), 391
 pulmonary circulation and alveolar ventilation perfusion relationships after (Cournand, Riley, Himmelstein, and Austrian), 80
 with contamination, sodium tetradecyl sulfate, azochloramide, and sulfanilamide mixture in prevention of empyema following (Rigdon and Brantigan), 319
- Pneumonolysis and paraffin plombage for chronic pulmonary disease, evaluation of extrapleural (Decker), 629
 with plombage, pulmonary function studies before and after extrapleural (Dressler, Bronfin, and Grow), 938
- Pneumothorax, surgical management of chronic "spontaneous" (Brewer, Dolley, and Evans), 167
 treatment of simple spontaneous (Curti and Poulsen), 145
- Postlethwait, R. W. (with Deaton, W. Ralph, Jr., Bradshaw, H. H., and Williams, Robert W.). Intying rubber tube after esophageal anastomosis, 649
- Potts-Smith-Gibson clamp for division of patent ductus arteriosus, use of (Conklin and Watkins), 361
- Poulsen, Thue (with Curti, P. C.). Treatment of simple spontaneous pneumothorax, 145
- Prietto, Carlos A. (with Shek, John L., Tuttle, W. M., and O'Brien, E. J.). Experimental study of blood supply of esophagus and its relation to esophageal resection and anastomosis, 523, 541
- Pulmonary alveolar adenomatosis; cancerous pulmonary adenomatosis, alveolar cell carcinoma of lung, Jag-ziekte? (Stephens and Shipman), 559
 carcinoma, intrapericardial anatomy in relation to pneumonectomy for (Healey and Gibbon), 864
 circulation and alveolar ventilation perfusion relationships after pneumonectomy (Cournand, Riley, Himmelstein, and Austrian), 80
 disease, evaluation of extrapleural pneumonolysis and paraffin plombage for chronic (Decker), 629
 function studies before and after extrapleural pneumonolysis with plombage (Dressler, Bronfin, and Grow), 938
 hydatid disease, treatment of (Susman), 422
 lesions (tuberculomas), surgical treatment of round tuberculous (Mahon and Forsee), 724
 resection for metastatic malignant lesions (Seiler, Clagett, and McDonald), 655
 vein and right diaphragm, bronchiectasis associated with anomaly of (Drake and Lynch), 433
- Puppel, I. Darin. Role of esophageal motility in surgical treatment of mega-esophagus, 371
- Purves, Robert K. (with Wedin, Paul H.). Familial incidence of cervical ribs, 952

R

- Rabin, Coleman B. (with Janowitz, Henry D.). Actinomyces in putrid empyema, 355
- Raine, Forrester. Discussion of funnel chest and deformities of thoracic cage, 519
- Ramsay, Beatty H. (with Overholt, Richard H., and Woods, Francis M.). Segmental pulmonary resection, 207
- Reganis, John C. (with Kisner, Wendell H.). Pericardial celomic cyst, with symptoms, 779
- Removal of lower accessory lung, successful (DeBakey, Arey, and Brunazzi), 304
- Resecting large areas of chest wall, use of tantalum plate when (Beardsley), 444

- Mega-esophagus, role of esophageal motility in surgical treatment of (Puppel), 371
- Merendino, K. Alvin (with Emerson, E. C.). Aortoesophagogastric fistula, unusual complication of esophagogastrostomy performed under aortic arch following esophageal resection for carcinoma, 405
- Merrill, Keith, Jr. (with Swenson, Orvar, Peirce, E. Converse, II, and Rheinlander, Harold F.). Blood and nerve supply to esophagus, 462
- Mesothelial mediastinal cysts (Drash and Hyer), 755
- Mesothelioma, pleural (Sano, Weiss, and Gault), 783
- Meyler, L. (with Huizinga, Eelco). Temporary high position of diaphragm, 283
- Miller, Bernard J. (with Allbritten, Frank F., Jr., Lipshutz, Herbert, and Gibson, John H., Jr.). Blood volume changes in tuberculous patients treated by thoracoplasty, 71
- Mitral stenosis, surgery of (Bailey, Glover, and O'Neill), 16
surgical treatment of. II. Progress in developing controlled valvuloplastic technique (Harken, Ellis, and Norman), 1
- Moen, Chester W. (with Meade, Richard H., Jr., and Head, Jerome R.). Management of chylothorax, 709
- Moore, Julian. Discussion of chronic "spontaneous" pneumothorax, 195
- Morton, Douglas R. (with Osborne, Johnston F., and Klassen, Karl P.). An apparently congenital bronchoesophageal fistula persistent to adult life, 811
- Mulvihill, D. A. Discussion of segmental resection, 222
- Munro, D. D. (with Baxter, S. G.). Benign fibroma of chest wall, 944
- Murphy, Anna J. (with Beecher, Henry K.). Acidosis during thoracic surgery, 50
- Murphy, J. D. Discussion of segmental resection, 223
- N
- Narodick, Benjamin G. (with Rothstein, Emil, and Landis, Francis B.). Bronchospirrometry in lateral decubitus position, 821
- Neuhof, Harold (with Osserman, Kermit E.). Mucocellular papillary adenocarcinoma of lung; lobectomy, five year follow-up, 875
- Norman, Leona R. (with Harken, Dwight E., and Ellis, Laurence B.). Surgical treatment of mitral stenosis. II. Progress in developing a controlled valvuloplastic technique, 1
- Norris, Charles M. (with Long, Joan H., Burnett, W. Emory, and Wester, Mary R.). Bronchospirrometric studies in bronchiectasis, before and after lobectomy, 477
- O
- O'Brien, E. J. Discussion of plastic reconstruction of tuberculous bronchostenosis with dermal grafts, 623, 624
(with Shek, John L., Prietto, Carlos A., and Tuttle, W. M.) Experimental study of blood supply of esophagus and its relation to esophageal resection and anastomoses, 523
- Ochsner, Alton. Discussion of funnel chest and deformities of thoracic cage, 518
- O'Neill, Thomas J. E. (with Bailey, Charles P., and Glover, Robert P.). Surgery of mitral stenosis, 16
- Osborne, Johnston F. (with Morton, Douglas R., and Klassen, Karl P.). An apparently congenital bronchoesophageal fistula persistent to adult life, 811
- Osserman, Kermit E. (with Neuhof, Harold). Mucocellular papillary adenocarcinoma of lung, lobectomy, five year follow-up, 875
- Overholt, Richard H. (with Woods, Francis M., and Ramsay, Beatty H.). Segmental pulmonary resection, 207
- P
- Paine, John R. Discussion of combined abdominal and right thoracic approach to lesions of middle and upper thirds of esophagus, 568
Discussion of symposium on resection of esophagus, 537
- Paraffin plombage for chronic pulmonary disease, evaluation of extrapleural pneumonolysis and (Decker), 629
- Parker, E. F. Discussion of resection of esophagus, 538
- Patent ductus arteriosus, use of Potts-Smith-Gibson clamp for division of (Conklin and Watkins), 361
- Paulson, Donald L. Discussion of mediastinal "tuberculoma," 347
- Peirce, E. Converse, II (with Swenson, Orvar, Merrill, Keith, Jr., and Rheinlander, Harold F.). Blood and nerve supply to esophagus, 462
- Pericardial celomic cyst, with symptoms (Kisner and Reganis), 779
- Phillips, Francis J. (with Basinger, Clair E., and Adams, William E.). Bronchiogenic carcinoma. I. Pathologic clinical correlative study of full-size mounts from operated carcinomas, 680, 698

- Spinal canal, chondrosarcoma of posterior mediastinum with hourglass involvement of; resection and recovery (Weisel and Ross), 643
- Staines, Enrique (with Cárdenas, Consuelo). Streptomycin plus alkalinization of pleural cavity in treatment of tuberculous empyema, 891
- Stemmermann, Grant N. (with Duffy, Robert W.). Accidental cyst of chest wall lined with bronchial epithelium, 349
- Stephens, H. Brodie (with Shipman, Sidney J.). Pulmonary alveolar adenomatosis; cancerous pulmonary adenomatosis, alveolar cell carcinoma of lung, Jagzlekte?, 589, 603
- Stewart, John S. Roentgenologic manifestation of parasternal omental hernia, 399
- Streptomycin in treatment of tuberculous empyema, thoracoplasty and (Connolly and Forsee), 877
- plus alkalinization of pleural cavity in treatment of tuberculous empyema (Staines and Cárdenas), 891
- Susman, M. P. Treatment of pulmonary hydatid disease, 422
- Suture materials, mechanism of healing of lung tissue and its reaction to different (Valle and Mider), 324
- Sweet, Richard. Discussion of combined abdominal and right thoracic approach to lesions of middle and upper thirds of esophagus, 567
- Discussion of further observations on packing of mediastinum for esophageal varices, 587
- Discussion of mitral stenosis, 45
- Discussion of resection of esophagus, 539
- Lobectomy and pneumonectomy in treatment of pulmonary tuberculosis, 298
- Swenson, Orvar (with Keith, Merrill, Jr., Peirce, E. Converse, II, and Rhinlander, Harold F.). Blood and nerve supply to esophagus, 462
- T
- Tantalum plate when resecting large areas of chest wall (Beardsley), 444
- Taylor, Selwyn (with Franklin, R. H.). Nonspecific granulomatous (regional) esophagitis, 292
- Temple, Leslie J. Aneurysm of first part of left subclavian artery, 412
- Thomas, Vivien (with Hanlon, C. Rollins, and Johns, Thomas N. P.). Apparatus for anesthesia in experimental thoracic surgery, 887
- Thoracic approach to lesions of middle and upper thirds of esophagus, combined abdominal and right (Kent and Harbison), 559
- cage, funnel chest and allied deformities of (Lester), 507
- Thoracic—Cont'd
- surgery, apparatus for anesthesia in experimental (Hanlon, Johns, and Thomas), 887
- wall, reconstruction of anterior (Campbell), 456
- Thoracoplasty and streptomycin in treatment of tuberculous empyema (Connolly and Forsee), 877
- blood volume changes in tuberculous patients treated by (Allbritton, Lutz, Miller, and Gibbon), 882
- during pregnancy, observations on (Intyre), 882
- evaluation of apicectomy in (Meinke), 833
- late results in four hundred twenty tuberculous patients subjected to (Meinke), 837
- pneumonectomy followed by (Iverson and Skinner), 451
- study of fifty-six consecutive (Shipman, Segal, and Stewart), 451
- transscapular (Coello), 509
- Thyroidectomy, transthoracic (Lester), 507
- Touroff, Arthur S. W. Discussion of "spontaneous" pneumothorax, 723
- Discussion of management of (Lester), 507

- Resection and anastomoses, an experimental study of blood supply of esophagus and its relation to esophageal (Shek, Prietto, Tuttle, and O'Brien), 523
- and end-to-end anastomosis, treatment of short esophageal strictures by (Tuttle and Day), 534
- for metastatic malignant lesions, pulmonary (Seiler, Clagett, and McDonald), 655
- in pulmonary diseases, segmental (Chamberlain and Ryan), 199
- of human carina, tracheal wall, and contralateral bronchial wall in cases of right total pneumonectomy, experiences with surgical (Abbott), 906
- segmental pulmonary (Overholt, Woods, and Ramsay), 207
- Respiration, electrophrenic. IV. Effectiveness of contralateral ventilation during activity of one phrenic nerve (Sarnoff, Gaensler, and Maloney), 929
- new method of automatic controlled (Adelman, Berman, and Touroff), 817
- Rheinlander, Harold F. (with Swenson, Orvar, Merrill, Keith, Jr., and Peirce, E. Converse, II). Blood and nerve supply to esophagus, 462
- Rigdon, H. L. (with Brantigan, O. C.). Sodium tetradecyl sulfate, azochloramide, and sulfanilamide mixture in prevention of empyema following pneumonectomy with contamination, 319
- Riley, R. L. (with Cournand, A., Himmelstein, A., and Austrian, R.). Pulmonary circulation and alveolar ventilation perfusion relationships after pneumonectomy, 80
- Roentgenologic manifestations of parasternal omental hernia (Stewart), 399
- Ross, Willard B. (with Weisel, Wilson). Chondrosarcoma of posterior mediastinum with hourglass involvement of spinal canal; resection and recovery, 643
- Rothstein, Emil (with Landis, Francis B., and Narodick, Benjamin G.). Bronchospirrometry in lateral decubitus position, 821
- Rumel, William R. Discussion of management of chylothorax, 721
- Ryan, Thomas C. (with Chamberlain, J. Maxwell). Segmental resection in pulmonary diseases, 199
- Rydell, John R. (with Grindlay, John H., and Clagett, O. Theron). Permanent filling of dead space in pleural cavity after pneumonectomy, 391
- S
- Samson, Paul C. Discussion of segmental resection, 220
- (with Heaton, Leonard D., and Dugan, David J.). Mediastinal "tuberculoma," 333, 348
- Sanger, Paul W. Discussion of pneumonectomy followed by immediate thoracoplasty, 505
- Sano, Machteld E. (with Weiss, Edward, and Gault, Edwin S.). Pleural mesothelioma, 783
- Sarnoff, Stanley J. (with Gaensler, Edward A., and Maloney, James V., Jr.). Electrophrenic respiration. IV. Effectiveness of contralateral ventilation during activity of one phrenic valve, 929
- Scannell, J. Gordon. Discussion of management of chylothorax, 720
- Schafer, Paul W. (with Kittle, C. Frederick, and Boley, James O.). Resection of an intrathoracic "hibernoma," 830
- Schmidt, Herbert W. (with Clagett, O. Theron, and McDonald, John R.). Broncholithiasis, 226, 245
- Segal, J. (with Goldman, Alfred, and Sherman, Leon H.). Thoracoplasty, 270
- Segmental pulmonary resection (Overholt, Woods, and Ramsay), 207
- resection in pulmonary diseases (Chamberlain and Ryan), 199
- Seiler, Hawley H. (with Clagett, O. Theron, and McDonald, John R.). Pulmonary resection for metastatic malignant lesions, 655
- Semb, Carl. Discussion of pneumonectomy followed by immediate thoracoplasty, 504, 506
- Shaw, Robert R. Discussion of segmental resection, 221
- Shek, John L. (with Prietto, Carlos A., Tuttle, W. M., and O'Brien, E. J.). Experimental study of blood supply of esophagus and its relation to esophageal resection and anastomoses, 523
- Sherman, Leon H. (with Goldman, Alfred, and Segal, J.). Thoracoplasty, 270
- Shipman, Sidney J. (with Stephens, H. Brodie). Pulmonary alveolar adenomatosis; cancerous pulmonary adenomatosis, alveolar cell carcinoma of lung, Jagziekte?, 589
- Skinner, George F. Discussion of mesothelial mediastinal cysts, 767
- Skinner, Homer L. (with Iverson, Roland K.). Pneumonectomy followed by immediate thoracoplasty, 491, 506
- Snyder, J. M. Discussion of further observations on packing of mediastinum for esophageal varices, 586
- Soave, Franco. Anesthetic blocking of vagus, sympathetic, and phrenic at neck in thoracic surgery—its protective action on blood pressure, 157
- Som, Max L. (with Garlock, John H.). Further observations on packing of mediastinum for esophageal varices, 572
- Sommer, G. N. J., Jr. Discussion of pulmonary resection for metastatic malignant lesions, 677

V

- Valle, Anibal Roberto (with Mider, G. Burroughs). Mechanism of healing of lung tissue and its reaction to different suture materials, 324
- Valvuloplastic technique, surgical treatment of mitral stenosis. II. Progress in developing a controlled (Harken, Ellis and Norman), 1
- Van Hazel, Willard. Discussion of pulmonary resection for metastatic malignant lesions, 678
- Ventilation during activity of one phrenic nerve, electrophrenic respiration. IV. Effectiveness of contralateral (Sarnoff, Gaensler, and Maloney), 929

W

- Waterman, D. H. Discussion of blood volume changes in tuberculous patients treated by thoracoplasty, 78
- Discussion of pulmonary resection for metastatic malignant lesions, 675
- Discussion of surgical treatment of bronchiectasis, 267
- Watkins, Elton, Jr. (with Conklin, William S.). Use of Potts-Smith-Gibson clamp for division of patent ductus arteriosus, 361
- Wedin, Paul H. (with Purves, Robert K.). Familial incidence of cervical ribs, 952

- Weinberg, Joseph A. (with Kraus, Alvin R.). Intercostal incision in trans-pleural operations, 769
- Weisel, Wilson (with Cassel, M. A., and Cunningham, R. T.). Foregut cyst of mediastinum, 138
- (with Ross, Willard B.). Chondrosarcoma of posterior mediastinum with hour-glass involvement of spinal canal; resection and recovery, 643
- Weiss, Edward (with Sano, Machteld E., and Gault, Edwin S.). Pleural mesothelioma, 783
- Wester, Mary R. (with Long, Joan H., Norris, Charles M., and Burnett, W. Emory). Bronchospirometric studies in bronchiectasis, before and after lobectomy, 477
- Williams, R. B., Jr. (with Daniel, Rollin A., Jr.). Leiomyoma of esophagus, 800
- (with Daniel, Rollin A., Jr.). Leiomyoma of lung, 806
- Williams, Robert W. (with Deaton, W. Ralph, Jr., Postlethwait, R. W., and Bradshaw, H. H.). Inlying rubber tube after esophageal anastomosis, 649
- Woodruff, Warriner. Discussion of mediastinal "tuberculoma," 348
- Woods, Francis M. Discussion of chronic "spontaneous" pneumothorax, 196
- Discussion of segmental resection, 224
- (with Overholt, Richard H., and Ramsay, Beatty H.). Segmental pulmonary resection, 207